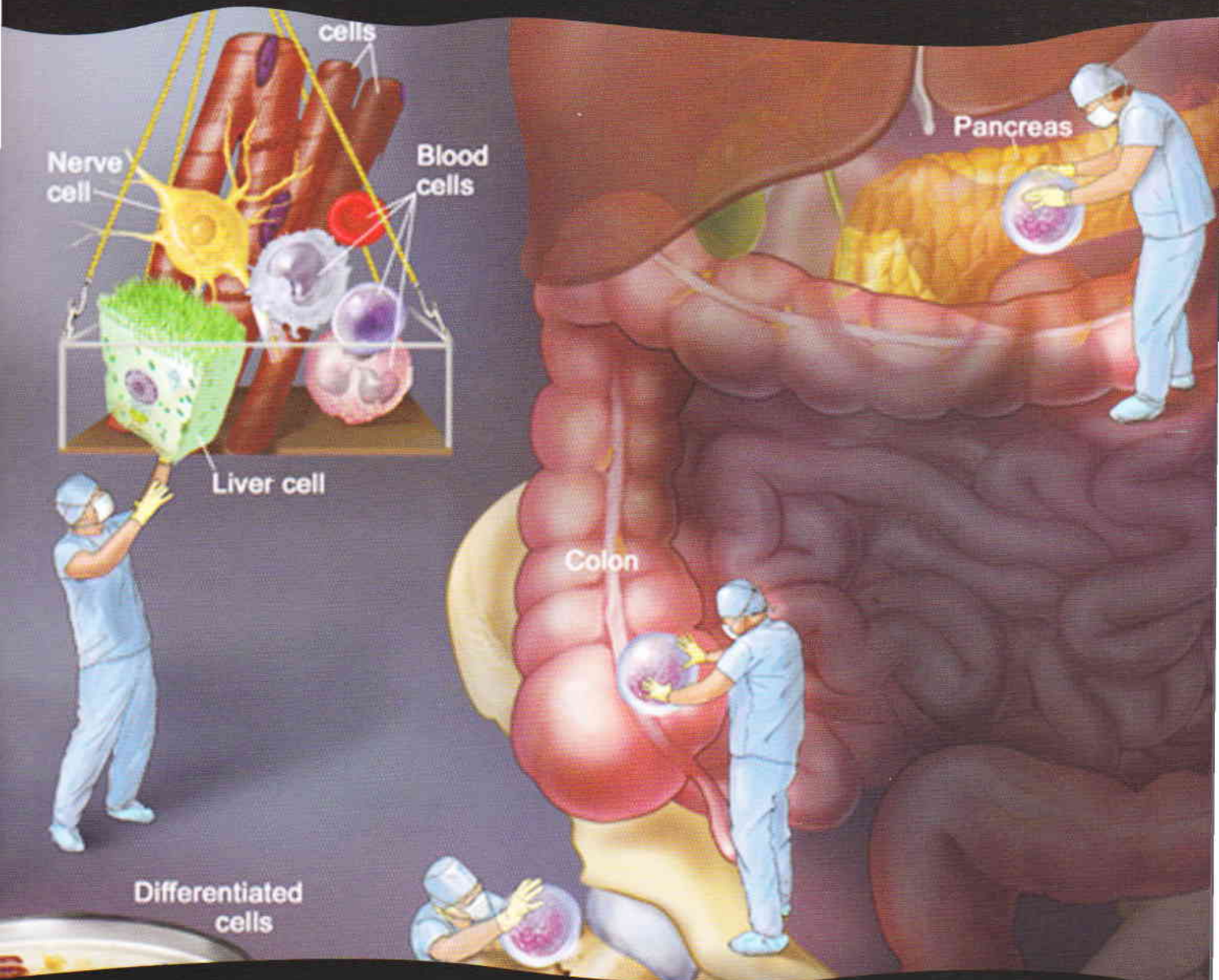


Matary Textbook of

GASTROINTESTINAL SURGERY

Mohammed El-Matary



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Dedication

Allah the all merciful, I beg Thee
To accept this effort
For the soul of my mother
She was your gift for me

Acknowledgement

The author wishes to acknowledge with gratitude:

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Preface

This book provides an update for medical students who need to keep abreast of recent developments. I hope also it will be useful for those preparing for postgraduate examination.

This book is designed to provide a concise summary of Gastrointestinal surgery, which medical students and others can use as study guide by itself or with readings in current textbooks, monographs, and reviews.

The author is extremely grateful to all the contributors for the high standard of the new chapters, and hopes that you, the reader, will enjoy going through these pages as much as he had.

M. El-Matary

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Abbreviations

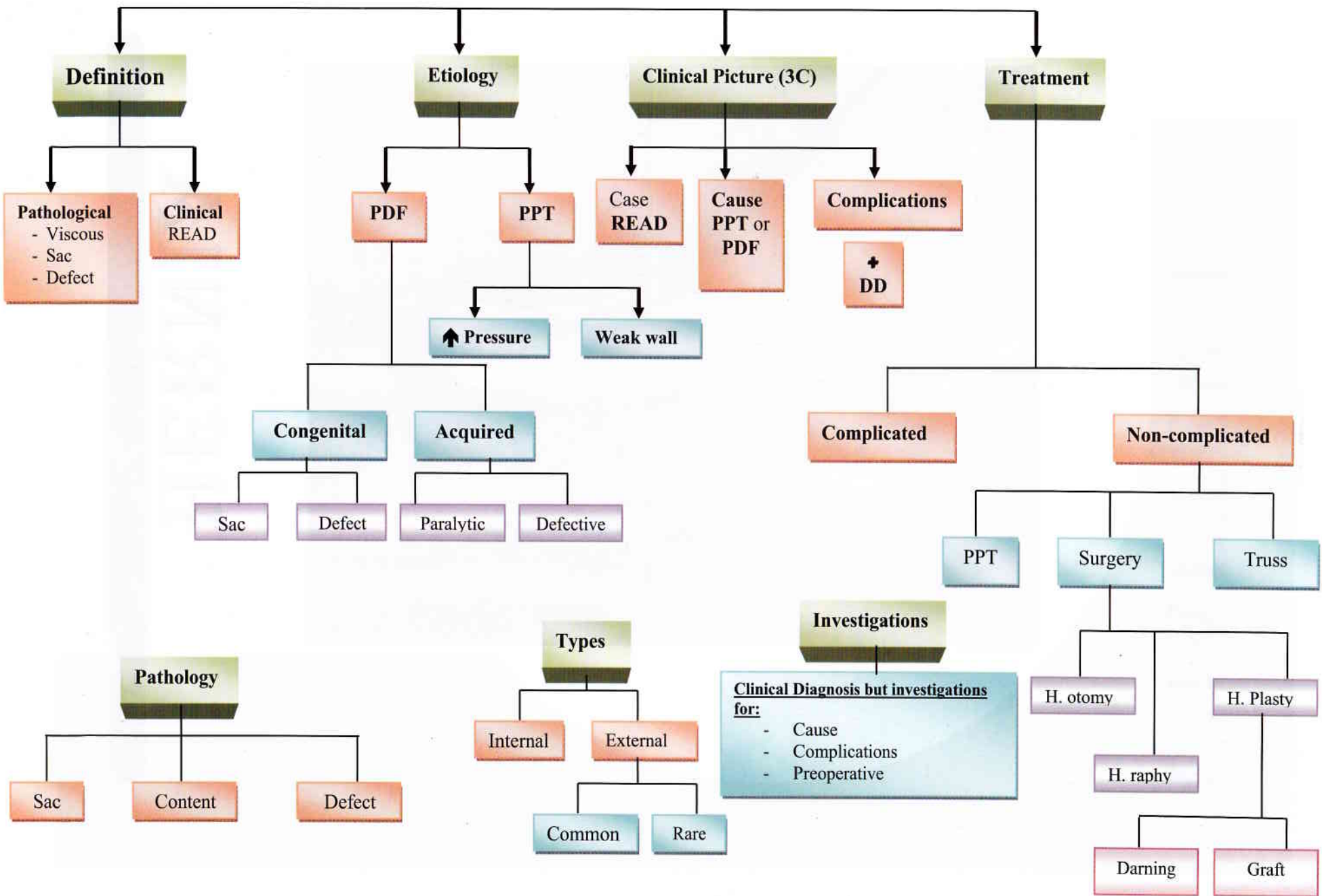
ACTH	Adrenocorticotrophic Hormone
AF	Atrial Fibrillation
ALP	Alkaline Phosphatase
ALT	Alanine transferase
AST	Aspartate transferase
A-V	Arterio-Venous
BP	Blood Pressure
BUN	Blood Urea & Nitrogen
C/P	Clinical Picture
CBC	Complete Blood Count
CBD	Common Bile Duct
CEA	Carcino Embryonic Antigen
CFT	Complement Fixation Test
CHD	Common Hepatic Duct
CHO	Carbohydrate
CMV	Cyto Megalo Virus
CNS	Central Nervous System
CT	Connective Tissue / Computerized Tomography
CUSA	Cavitron Ultrasonic Surgical Aspirator
CVS	Cardio Vascular System
DD	Deffrential Diagnosis
DKA	Diabetic Keto acidosis
DM	Diabetes Mellitus
DNA	Deoxyribonucleic Acid
DPL	Diagnostic Peritoneal Lavage
DRE	Digital Rectal Examination
DSA	Digital Subtraction Angiography
DVT	Deep Venous Thrombosis
EBV	Ebstein Barr Virus
ECG	Electro Cardiography
EEA	End-to-End American stapler
ERCP	Endoscopic Retrograde Colangio Pancreatography
ESR	Erythrocytic Sedimentation Rate
FNH	Focal Nodular Hyperplasia
FPC	Familial Polyposis Coli
FUO	Fever of Unknown Origin
GB	Gall Bladder
GIT	Gastr Intestinal Tract
HCC	Hepato Cellular Carcinoma
HIDA	Hydroxy Imedinodiacetic Acid
HSD	Hirschsprung's Disease
HSV	Herpes Simplex Virus
HTN	Hypertension
IBD	Inflammatory Bowel Disease

IHA	Indirect Heme Agglutination
IM	Intra Muscular
IO	Intestinal Obstruction
ITP	Idiopathic Thrombocytopenic Purpura
IU	International Unit
IV	Intra Venous
IVC	Inferior Vena Cava
IVP	Intra Venous Pyelography
KFTs	Kidney Function Tests
LCF	Liver Cell failure
LFTs	Liver Function Tests
LN	Lymph Nodes
MI	Mitral Incompetence
MRI	Magnetic Resonance Imaging
MS	Mitral Stenosis
MVO	Mesenteric Vascular Occlusion
NG	Naso Gastric
PMN	Polymorph Neutrophils
PTC	Percutaneous Transhepatic Cholangiopancreatography
PTH	Parathyroid Hormone
PU	Peptic Ulcer
RES	Reticulo Endothelial System
RP	Rectal Prolapse
RVF	Right Ventricular Failure
SCC	Squamous Cell Carcinoma
SGOT	Serum Glutamate Oxaloacetate Transaminase
SGPT	Serum Glutamate Pyruvate Transaminase
SI	small Intestine
SMA	Superior Mesenteric Artery
SMV	Superior Mesenteric Vein
STD	Sexually Transmitted Disease
TB	Tuberculosis
TCA	Tricyclic Antidepressant
tds	3 times per day
TIPSS	Transjugular Intrahepatic Porto-Systemic Shunting
TLC	Total Leukocytic Counting
ttt	Treatment
U/S	Ultrasonography
UB	Urinary Bladder
UC	Ulcerative Colitis
VD	Vasodilatation / Vasodilator

CHAPTER 1



HERNIA



General Schemes of Abdominal Hernias

What is hernia?

- Protrusion of organ from its cavity through a defect.

Definition of abdominal hernia

⇒ Pathologically:

- Hernia is a protrusion of a **viscus** or a part of viscus, usually within a peritoneal **sac** through a congenital or acquired **defect** in the abdominal wall.

⇒ Clinically: (Painless swelling characterized by **READ**)

- 1- Reducible or gives history of reducibility.
- 2- Gives **Expansile** impulse on cough.
- 3- On the **Anatomical** site of hernia.
- 4- With **Defect**.

Etiology

A- Predisposing factors

1. Congenital:

- a) Congenital sac (unobliterated processus vaginalis): → congenital inguinal hernia.
- b) Congenital defect (unobliterated physiological umbilical hernia): → congenital umbilical hernia (exomphalos).

2. Acquired: (incisional hernia)

a) Paralytic type:

1. Grid iron incision with Rutherford Morison extension.
 - Injury of ileo-inguinal nerve (supplying conjoint tendon) → direct inguinal hernia
2. Kocher's incision → Paralysis of intercostal n.

b) Defective type:

- Due to absorbable suture material or loose sutures

B- Precipitating factors

1. Raised intra-abdominal pressure (precipitating factors) due to:

- Chronic cough.
- Straining due to constipation, prostatism.
- Obesity.
- Abdominal swelling (Splenomegaly).

2. Weak anterior abdominal wall due to:

- Repeated pregnancy.
- Obesity.
- Senility.

Pathology

- A hernia consists of three parts which are the **sac**, the **content** and the coverings of the sac. The content passes out through a **defect** in the abdominal wall.

A- Defect

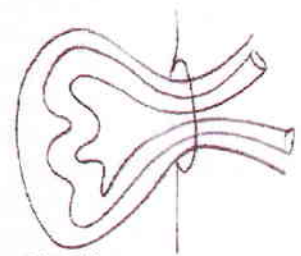
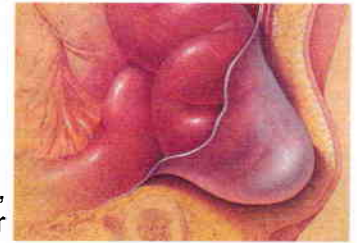
- Either congenital or acquired.

B- Sac

- A peritoneal pouch that bulges out through abdominal wall defect.
- It has a neck, body & fundus and may be adherent to the coverings.

▪ Neck is:

- Wide except in femoral & para-umbilical hernias.
- Well defined except in incisional & direct hernias.



pathology of hernia

C- Contents

Fluid is considered the commonest content.

1. Omentum → it gives doughy sensation (omentocoele = epiplocele).
 2. Intestine → soft, gurgle, resonant (enterocoele).
 3. Fluid → Hydrocele of hernial sac.
 4. Meckel's diverticulum → Littre's hernia.
- The usual contents are intestine, omentum or both:

	Intestine	Omentum
Consistency	Soft	Doughy
Gurgling	+ve	- ve
Reduction	difficult at 1 st then easy	Easy at 1 st then difficult
Percussion!!	Resonant	Dull
X ray	Gases	No gases

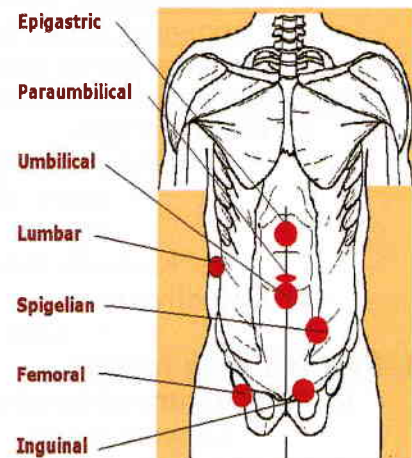
- **N. B.** Any abdominal viscus can be content except **Pancreas**

➤ **Why?**

- It is retroperitoneal
- It has a tail.
- Behind it → Aorta, IVC & vertebra.

Classification of hernia**A. According to site**

1. Internal (e.g. Hiatus hernia).
2. External (direct & indirect inguinal hernia).

**B. According to incidence**a) **Common:**

1. Inguinal region (most common, may be indirect, 65% or direct, 35%).
2. Incisional (post-operative hernia) → 2nd most common.
3. Epigastric region.
4. Femoral region.
5. Umbilical region: may be umbilical or paraumbilical hernia.

b) **Rare types:**

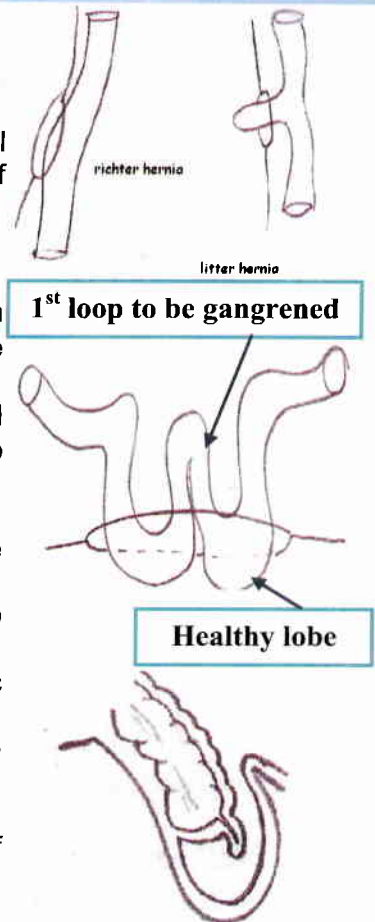
- **Obturator hernia:** through the obturator foramen.
- **Lumbar hernia:** through the lumbar triangles.
- **Gluteal hernia:** through the greater sciatic foramen.
- **Sciatic hernia:** through the lesser sciatic foramen.
- **Spigelian hernia:** through the linea semilunaris.
- **Perineal hernia:** into the perineum

Hernia

5

Special types of hernia

- ❑ **Littre's hernia:** the content is Meckel's diverticulum.
- ❑ **Richter's hernia:**
 - The content is a part of the circumference of an intestinal loop (common in femoral hernia), diagnosis only if strangulated.
 - It causes strangulation without obstruction (**patent lumen**)
- ❑ **Maydl's (W) hernia:**
 - The content is 2 loops of intestine with intermediate loop in the peritoneal cavity (if **strangulated, sac may not be tender**).
 - So, whenever we find the W-shaped hernia we have to pull over the medial limb (which may be strangulated) to convert it into U-shaped.
- ❑ **Sliding hernia:**
 - Urinary bladder, caecum or colon passes through the defect adherent to the sac.
 - Gives partial irreducibility and predisposes to complications.
- ❑ **Pantaloon (dual hernia):** 2 sacs, with inferior epigastric artery in between.
- ❑ **Interstitial hernia:** the sac passes between the layers of the abdominal wall.
 - **Diagnosis:** usually by CT.
 - **TTT:** by either mesh repair or the "separation of components" technique to obliterate the defect.



Clinical picture of external hernia

A- Clinical characters (Case)

"Painless swelling"

1. **Reducible** except if complicated, or gives history of reducibility.
2. Gives **expansile impulse** on cough except if strangulated.
3. On the **anatomical site** of hernia.
4. By **trans-illumination** it is opaque except in infants.

B- Clinical picture of complications

C- History of precipitating factor (Cause)

D- DD

Investigations

Hernia is a clinical diagnosis

- **Investigations are done to detect precipitating factors, complications and as preoperative preparation of all patients.**

1. CXR.
2. Abdominal & trans-rectal U/S (for BPH in old age).
3. CBC, FBS & ECG.
4. Liver function tests & albumin.
5. Kidney function tests.

- **In occult, unusual or doubtful cases US, CT or Laparoscope may be required for diagnosis.**



Typical picture of hernia Note: anatomical site of hernia & reducible

Treatment

A- Non- complicated cases

- Avoidance & treatment of **precipitating factors**.
 - **Surgery** is always advised to avoid the complications of hernia.
 - **Truss** is better to be avoided (as it may precipitate strangulation).
- **Truss may be used for extremely ill, unfit patients with reducible hernia**

1. Herniotomy:

- It includes excision of the hernial sac from the neck after reduction of the contents.
 - It is either done:
 - Alone in case of congenital inguinal hernia in child.
 - With herniorrhaphy or hernioplasty in adulthood.

2. Herniorrhaphy:

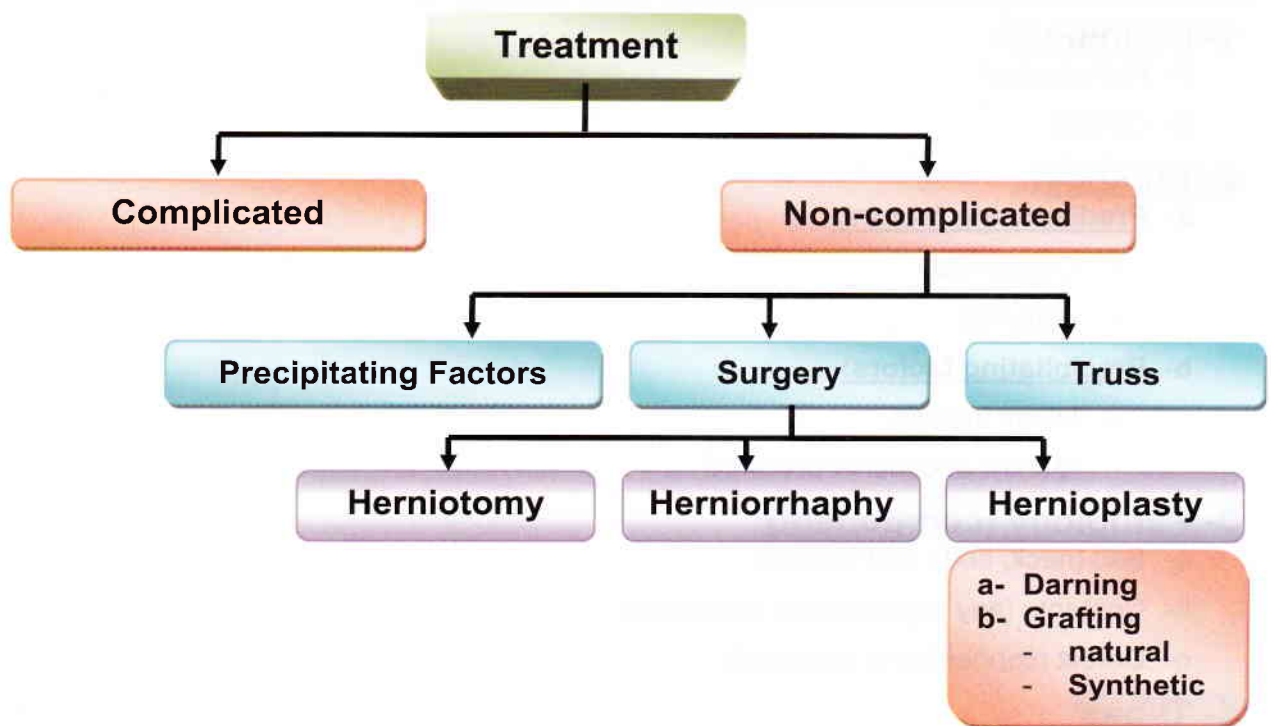
- It includes herniotomy & repair of the hernial defect by:
 - a. Narrowing of hernial orifice (or close it completely).
 - b. Strengthening the abdominal wall by suture material
- It's indicated in cases with:
 - a. Small or moderate sized defect.
 - b. Moderate strength of abdominal muscles.
- Suture material used is prolene which is:
 - a. Synthetic
 - b. Non absorbable
 - c. Inert
 - d. Formed of one filament only (less liability to infection)
- Disadvantages include:
 - Tension → tissue strangulation (↓ vascularity) → Recurrence.

3. Hernioplasty:

- It includes herniotomy & closure of the defect by:
 - Darning.
 - Graft → Natural: by fascia lata or skin (**not done now**).
→ Synthetic: (proline, mersilene, PTFE).
- It's indicated in cases with:
 - a. Large sized defect.
 - b. Old age with weak abdominal muscles.
- Advantages:
 - No tension sutures → less recurrence rate.
- Disadvantage: as the mesh is a foreign body, if infected → we can't eradicate. Thus it's contra-indicated in case of strangulated hernia (potentially infected field).
- Application of mesh:
 - a- **In lay** → Mesh is put between peritoneum & fascia transversalis.
→ Good approach.
 - b- **Sub lay** → Mesh is put deep to peritoneum.
→ May cause intestinal adhesions.
 - c- **On lay** → Mesh is put in front of external oblique.
→ May cause infection.
- Technique of hernioplasty
 - a. The Lichtenstein tension-free hernia repair.
 - b. **Laparoscopic repair** (most recent) either a **totally extraperitoneal** (TEP) or a trans-abdominal pre-peritoneal (TAPP) approach.

B- Complicated cases

- See Complications.

**Weak abdominal muscle present by either:**

1. Multiplicity of hernias.
2. Divarication of recti.
3. Bulge of lower abdomen on straining (malgaigne's bulge).

Summary of General Scheme of any Hernia

1- Definition

- A- Pathological
- B- Clinical

2- Etiology

a- Predisposing factors:

- Congenital
- Acquired

b- Precipitating factors:

- Weak muscle
- ↑ intra-abdominal pressure

3- Pathology (component)

- a- Sac (neck, body and fundus)
- b- Contents (any organ except pancreas)
- c- Defect (congenital or acquired)

4- Types

- a- External (common, rare)
- b- Internal

5- Clinical Picture (3 C + DD)

- a. Cause
- b. Case → (R E A D) → **R**educible, **E**xpansile, **A**natomical site, **D**efect)
- c. Complications
- d. DD

6- Investigations

- a- It is a clinical diagnosis
- b- Preoperative investigations

7- Treatment

a- Non-complicated:

- TTT of cause
- Surgery (herniorrhaphy, herniotomy and hernioplasty)
- Palliative (Truss)

b- Complicated:

Complications of Hernia

- 1- Irreducibility
- 2- Obstruction
- 3- Inflammation
- 4- Strangulation
- 5- Hydrocele of hernia
- 6- Rupture of hernial sac
- 7- Torsion of omentum
- 8- Recurrence
- 9- Sliding

1- Irreducibility

- Failure of the whole hernial contents or a part of it to return to abdomen.

Causes

1. Adhesion between the sac & the contents (the commonest).
2. Adhesions between the contents.
3. Overcrowding of the contents.
4. Narrow neck (relative).
5. Sliding hernia (because the viscus is adherent to the sac).
6. Omentum in the sac → more fat content → less mobility → more adhesions.
→ Rough.
→ Bulky.

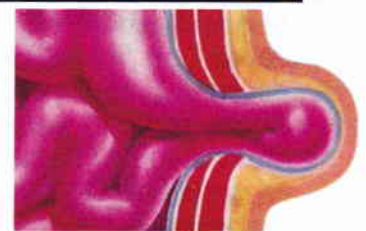
Treatment

- Surgical repair as early as possible (but not as urgent as strangulation).

Using a truss in irreducible hernia is contraindicated as it might lead to strangulation.

2- Obstruction (No interference with blood supply)

- Occlusion of intestinal lumen.
- It occurs usually in irreducible hernia.
- The intestinal lumen is obstructed from inside by faecolith (**incarcerated hernia**) or from outside (band of adhesion)
- There is no interference with its blood supply.



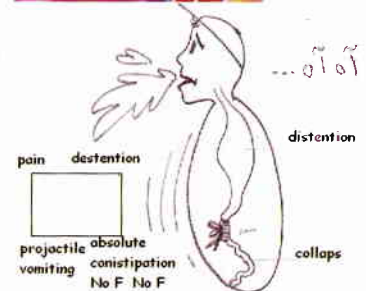
Clinical Picture

Symptoms (of intestinal obstruction)

- Abdominal colic, projectile vomiting, absolute constipation & distension.

Signs

- The hernial sac:
 - Slightly tender.
 - Soft (not tense).
 - Weak expansile impulse on cough (as it is large).
- The differentiation between obstruction and strangulation is difficult clinically, so they have the same treatment.



Treatment

- Urgent operation to avoid strangulation, because we can't differentiate between both.

3- Inflammation

Definition

- It is inflammation of the



Clinical Picture

Symptoms

➤ General:

- Fever, headache, anorexia and malaise.

➤ Local:

- Pain at the swelling
- Pain at McBurney's point. (inflamed appendix)

Signs

- The hernia is red, hot & tender.
- There is expansile impulse on cough.

Treatment

Treatment of the cause + hernia

- If due to truss → remove the truss + NSAIDs + elective hernia repair.
- If appendicitis → appendicectomy + hernia repair at the same session.

4- Strangulation (interference with blood supply)

Definition

- Interference with the blood supply of the contents with impending gangrene may occur within 4 - 6 hours.

Incidence

- Strangulated hernia is the commonest cause of intestinal obstruction in developing countries.
- Adults are more liable than infants (because of stronger muscles).
- Inguinal hernia is the commonest strangulated hernia (femoral hernia is the most liable to strangulation due to narrow neck, but it's less common than inguinal hernia, followed by umbilical hernia).
- The commonest contents to be strangulated are **small intestine & omentum** respectively.

- **Incidence:** ing.hernia is the commonest
- **Etiology:** sharp edge, narrow neck, other comp.
- **Symp.:** painful swelling, int. obstruction
- **Signs:** irreducible, tense, no impulse
- **TTT:** preparation + mainly resection & anastomosis

Causes

1. Sharp edge of the defect:

- Edge of the internal or external ring in oblique inguinal hernia.
- Edge of lacunar ligament in femoral hernia.
- Defect of the linea Alba in para- umbilical hernia.

2. Narrow neck: in relation to large contents.

3. Irreducibility, inflammation, obstruction predispose for strangulation (common after use of truss).

Pathology

1. The constricting agent:

- Any resistant structure outside sac.
- Bands of adhesions within sac.

2. The contents:

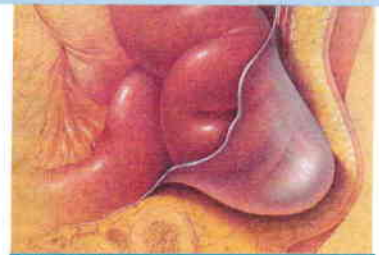
- Venous constriction → congestion of intestine → serous transudate.
- Arterial obstruction → ischemia of intestine → gangrene (starts at ring of constriction) → bloody exudates.

- Retrograde thrombosis might occur in the vessel till the first branch.
- Peritonitis is a terminal event as infection spread from the sac to the peritoneum. Neglected cases die from septic shock and dehydration.

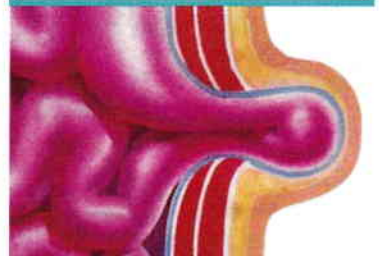
3. The sac:

- Distended with fluids.
- Loses its luster.

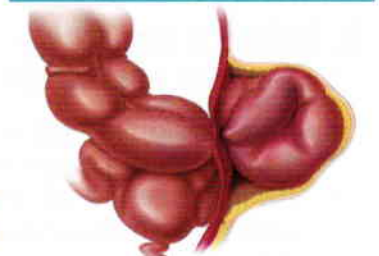
4. The covering: Inflamed with edema and congestion.



Uncomplicated



Obstructed



Strangulated

Clinical Picture

Pain + Tense + No impulse

➤ **Hernia is a painless condition unless complicated**

1. History of painless swelling that becomes painful:

- Type of pain → colicky & stabbing (due to ischemia) which is a cardinal symptom.
- Colics will disappear after perforation → generalized abdominal pain (peritonitis) finally septic shock.

2. Picture of intestinal obstruction:

- Repeated projectile vomiting, absolute constipation & distension.

3. Strangulation without obstruction may occur in:

1. Strangulated omentum.
2. Strangulated Richter's hernia.
3. Strangulated Meckel's diverticulum.

O/E:

▪ General:

- Bad general condition & **shock** (hypovolemic → due to fluid loss or neurogenic → due to severity of pain) (progress of shock depends on the extent of the affected loop).
- Abdominal distension, peritonitis when perforation occurs.

▪ Local: (the hernia)

- a- Gives no expansile impulse on cough (closed defect).
- b- Irreducible.
- c- **Tense** (full of exudate or transudate) & tender.
- d- **Sudden enlargement**

Treatment

Urgent operation after pre-operative preparation

A. Pre-operative preparation

1. Resuscitation (Mandatory):

⇒ GIT suction by Ryle's tube to:

1. Prevent vomiting.
2. Assess the amount of fluid lost (for fluid replacement).
3. Aspiration of toxins.
4. Prevent postoperative paralytic ileus by decreasing distention.
5. Prevent colic.
6. Prevent aspiration with induction of anesthesia.

⇒ Cannula for:

- IV fluid (ringer or saline).
- Blood transfusion in massive strangulation.
- Pre-operative medications (Morphia & Antibiotics).

⇒ Catheter for:

- Detection of urine output / hour to guard against pre-renal failure.

2. Monitoring:

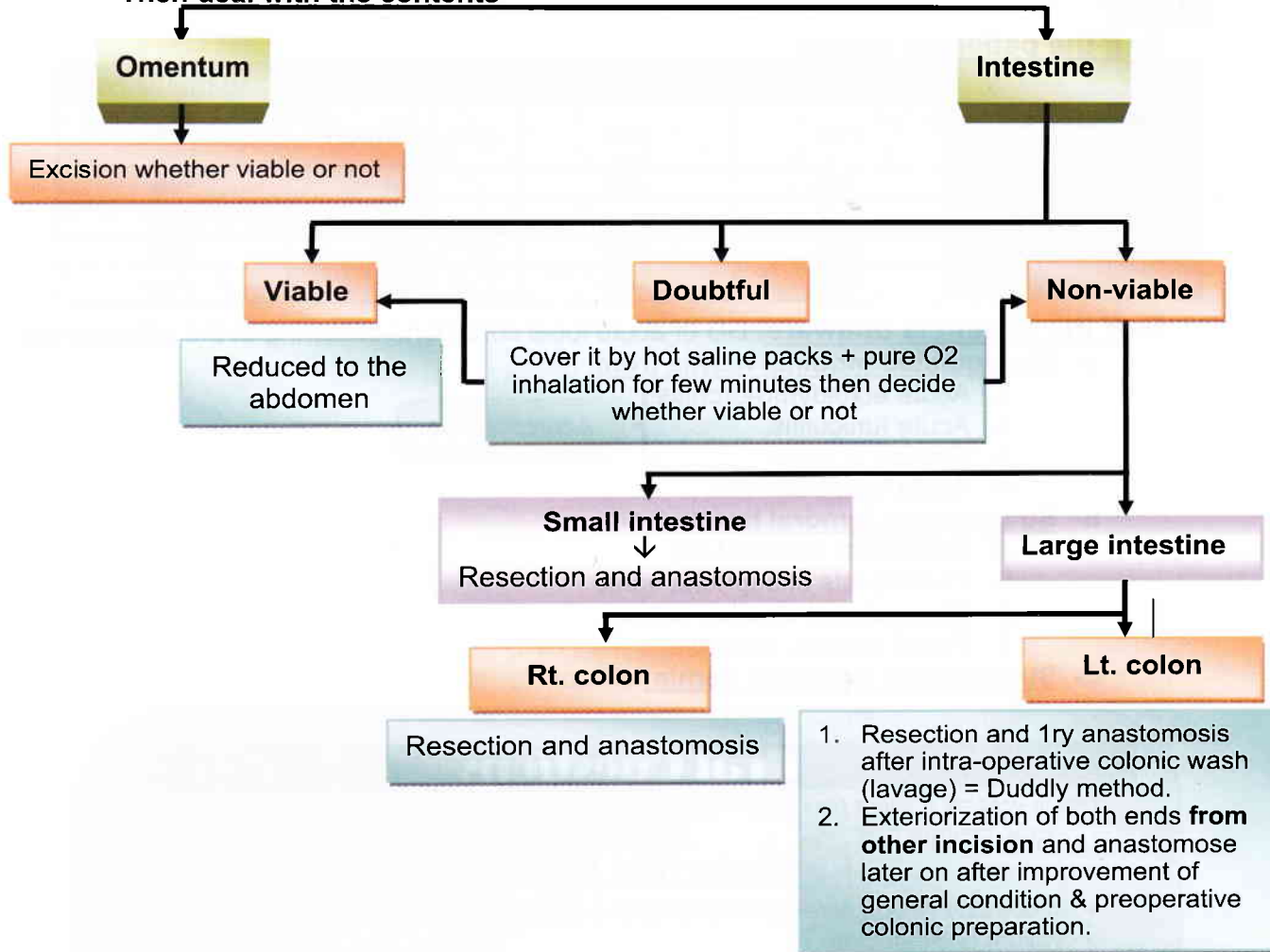
- Pulse, temperature, blood pressure and urine output.

B. Rules of operation

- General anesthesia (avoid spinal anesthesia → increases risk of post-operative paralytic ileus).
- The incision is wide & exploratory.
- Coverings are dissected to expose the sac.
- Sac is opened at the fundus (in inguinal & femoral hernia) or near its neck (in umbilical & paraumbilical hernia, because it is multi-loculated).
- Toxic fluid content is sucked out before division of the defect to avoid pouring of toxic contents into the circulation.
- Pulling loops of bowel so as not to miss strangulated Maydl's hernia
- Loop of the intestine is grasped gently by gauze to avoid slippage into the abdomen.
- Cut the constricting agent under vision over a hernia director or index finger.
- **If the contents are:**

	Viable intestine	Non-viable intestine
A. By inspection		
1- Color	Pink or dark red	Brown or black
2- Peritoneal luster	Present	Absent
B. By palpation		
1- Mesenteric artery	Pulsating	Non-pulsating
2- By pinching	Contracts	No response
3- Consistency	Firm with good tone	Floppy soft with no tone
4- If injures	Bleeding occurs	No bleeding
5- Peristalsis	Present	Absent
6- Elasticity	Present	Absent
7- Fluids in sac	At 1 st clear, later blood stained	May be stained or muddy and has feculent odour

- Then deal with the contents



- Excise the sac (herniotomy) & herniorrhaphy (**no hernioplasty as the tissues are potentially infected**).
- Closure in layers.
- Post-operative care:
 - Sedation
 - NPO.
 - Continue Ryle's suction with IV fluids.
 - Prophylactic antibiotics.
 - Drains are removed when discharging stop.

Special types of strangulated hernia

- Maydl's hernia (W- hernia)**
The gangrenous loop is found in the abdomen as there is double loop in the hernial sac.
- Richter's strangulated hernia**
Which is gangrene without obstruction as there is gangrene of the anti-mesenteric border of the intestine

DD

A- If the patient is aware:

	Irreducible	Inflamed	Obstructed	Strangulated
Impulse on cough	+ve	+ve	+ve (difficult)	-ve
Reducibility	-ve	-ve	-ve	-ve
A.I.O	-ve	-ve	+ve	+ve
Tender	-ve	+ve	+ve	+ve
Tense	-ve	-ve	-ve	+ve

B- If the patient is unaware: DD of acute local conditions occurring at the affected site:**a- Strangulated inguinal hernia from:**

- 1- Acute epididymo-orchitis.
- 2- Acute funiculitis.
- 3- Torsion of testis.
- 4- Acute lymphadenitis.

Acute scrotum

b- Strangulated femoral hernia from:

- 1- Adenitis of femoral LNs.
- 2- Thrombosis of saphena varix.
- 3- Strangulated inguinal hernia.
- 4- Psoas bursitis, abscess.

c- Strangulated umbilical hernia: difficult**Oral Questions***How to prepare a colon for closure of colostomy??*

Low residue diet

- enema (colostomy)

- Flagyl / neomycin

Reduction by taxis

- * Is done by flexion, internal rotation of the thigh to relax the external oblique.
- * Valium or Pethedine.
- * Cold compresses.
- * Trial of reduction after ½ an hour.
- * It is more useful in children with early strangulation.
- * Reduction by taxis (external reduction) is contra-indicated because:
 - o May cause shock.
 - o May cause rupture of the gut.
 - o Perforation in trial of reduction.
 - o Reduction en bisac into an intra-parietal sac.
 - o Reduction en mass.
 - o Reduction of gangrenous loop.

5- Hydrocele of the Hernial Sac

- It is due to reduction of the contents of the sac.
- The omentum obstructs the opening of the sac.
- Only fluid to pass to the sac.
- **Clinical picture:**
 - Cystic translucent inguinoscrotal swelling.
- **Treatment:** excision.

6- Rupture of Hernia Sac

- Due to external trauma as rough taxis

7- Torsion of the Omentum

- If the content is an omentum may twist upon itself → occlusion of the blood supply → gangrene.

8- Recurrence

- Will be discussed later at page 38.

9- Sliding

- Will be discussed later at page 27.

Inguinal Hernia

Anatomy

Layers of the anterior abdominal wall

- 1- Skin
- 2- Superficial fascia
- 3- Muscles

1. Skin

2. Superficial fascia:

- It's composed of superficial fatty layer (**Camper's fascia**) & deep membranous layer (**Scarpa's fascia**).

N.B. There's no deep fascia in the abdomen to allow free respiratory movement, gastric fullness & pregnancy.

➔ Fascia of Scarpa, Camper & Colle:

- Midway between the umbilicus and symphysis pubis.
- The deep part of the superficial fascia becomes condensed to form membranous layer called **Scarpa's fascia**.
- Scarpa's fascia ends below as follow:
 - It goes around penis, scrotum & perineum; called **Colle's fascia**.
 - More laterally join the **fascia lata** of the thigh.
- The fat superficial to Scarpa's fascia is called **Camper's fascia**. It loses its fat around the penis & scrotum (therefore, no lipoma in penis or scrotum).

3. Muscles:

1. External oblique muscle:

- **Origin:** lower 8 ribs.
- **Insertion:**
 - Fleshy → ant ½ of the outer lip of iliac crest.
 - Aponeurosis →
 - Medial: linea Alba, symphysis pubis, pubic crest, pubic tubercle.
 - Lateral: folded to form "Inguinal ligament".
- **Nerve supply:** Lower 6 thoracic nerves.
- **Direction:** downward, forward and medially.

2. Internal oblique muscle:

- **Origin:**
 - Lateral 2/3 of the upper surface of the inguinal ligament.
 - Anterior 2/3 of intermediate area of the iliac crest.
 - Lumbar fascia.
- **Insertion :**
 - Fleshy → lower 3 ribs.
 - Aponeurtoic →
 - Upper part
 - Middle part: linea alba.
 - Lower part: conjoint tendon.
- **Direction:** upward, forward and medially.

3. Transversus abdominis muscle:

- **Origin:**
 - Lateral 1/3 of the upper surface of the inguinal ligament.
 - Anterior 2/3 of the inner lip of the iliac crest.
 - Lumbar fascia.
 - Deep surface of the costal cartilage of the lower 6 ribs.
- **Insertion**
 - Linea alba.
 - Conjoint tendon.
- **Nerve supply:**
 - Lower 6 thoracic nerves.
 - First lumbar nerves.

➤ **Conjoint tendon:**

- **Fusion of** the lower parts of aponeurosis of internal oblique & transversus abdominis.
- **Inserted into:** • Pectin pubis • Pubic crest.
- **It forms** the medial part of the posterior wall of the inguinal canal just behind the superficial ring.

4. The transversalis “endo-abdominal” fascia:

- Connective tissue layer underlying the anterior abdominal wall muscles.
- Its lower part is thickened to form ilio-pubic tract.
- It extends downward as the anterior layer of femoral sheath and prolongs medially as internal cremasteric fascia.

5. Peritoneum.

6. The pre-peritoneal space:

- **Contents:**
 - Adipose tissue.
 - Lymphatics.
 - Nerves.
 - Inferior epigastric vessels (before entering rectus sheath).
 - Vas deferens (before joining the spermatic cord at int. ring).

*External oblique**Internal oblique**Transversus abdominis*

Inguinal canal (Deep inguinal pouch)

- This canal is formed by passage of testis from abdomen to scrotum.
- In adults, it's about 1.5 inches (4 cm) long.
- It extends downward, forwards & medially above the medial 1/2 of inguinal ligament

- Definition
- Begins
- Ends
- Wall
- Contents
- Hasselbach's triangle

Begins

⇒ At the internal ring:

- Opening in the fascia transversalis
- It lies 1/2 inch above the midpoint of inguinal ligament.
- It's an **inlet** for **spermatic cord** into the canal.
- It's supported **anteriorly** by **lower border of internal oblique**
- Inferior epigastric vessels run medial to it.

Ends

⇒ At the external ring:

- Opening in external oblique aponeurosis.
- It lies 1/2 inch above & medial to the pubic tubercle.
- It's an **exit** for **spermatic cord & ilioinguinal nerve**.
- It's supported **posteriorly** by **conjoint tendon**.
- Normally it doesn't admit the tip of the little finger.

The walls of the inguinal canal

1. Ant. Wall:

- External oblique.
- Lower lateral part of internal oblique.

2. Post. Wall:

- Fascia transversalis.
- Conjoint tendon (Medially).

3. Roof: arching fibers (internal oblique & transversus abdominis).

4. Floor: infolded surface of inguinal ligament (+ upper surface of lacunar ligament medially).

Contents

- Spermatic Cord (or round ligament in females).
- Ilio-inguinal nerve.

Triangle of Halshted (Hasselbach's or inguinal triangle)

It's the medial part of the posterior wall of the inguinal canal.

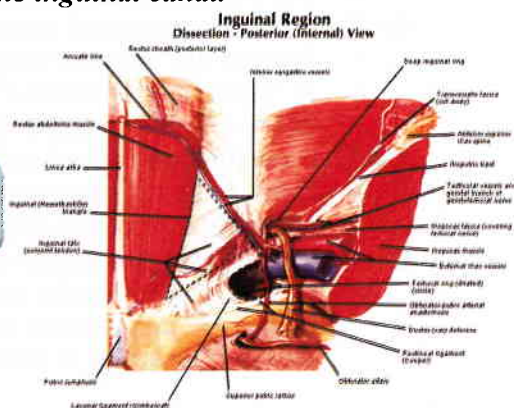
Its boundaries are

- **Laterally** → Inferior epigastric artery.
- **Medially** → Lateral border of rectus sheath.
- **Inferior** → Inguinal ligament (Med 1/2 of it).

- The triangle is further subdivided by the **lateral umbilical ligament** into medial & lateral parts.

Surgical importance

- Triangle through which direct hernia passes.



Spermatic cord (3C)

Course

- The cord begins at the deep inguinal ring by gathering of its contents.
- It enters through the deep ring into the inguinal canal, traverses it & exits through superficial ring.
- Then, it passes down in front of pubic bone, crossing the scrotal neck down to the scrotum where it is attached to the back of the testis.

Contents

3 Arteries	3 Nerves	3 Structures
1. Testicular a.	1. Ilioinguinal n.	1. Pampiniform plexus of veins
2. Cremasteric a.	2. Genital br. of genitofemoral n.	2. Vas deferens
3. Artery of vas	3. Sympathetic ns.	3. Obliterated processus vaginalis (Vestige).

Coverings

- ➔ The spermatic cord has three coverings which are derived from the penetrated layers of the abdominal wall during testicular descent.
 1. **Internal spermatic fascia** → from fascia transversalis (*at the deep ring*).
 2. **Cremasteric muscles** → from lower border of internal oblique (overlying deep ring).
 3. **External spermatic fascia** → from external oblique aponeurosis (at superficial ring).
- Ilioinguinal n. supplies conjoint tendon, while genital branch of genitofemoral supplies cremasteric muscle.

↪ Inguinal region is a weak spot: (surgical importance of inguinal region)

1. Muscles of abdominal wall are aponeurotic in inguinal region.
2. Internal oblique & transversus abdominis muscles arch up to form the roof of inguinal canal.
3. Spermatic cord passes between muscles & adds more weakness to inguinal area.

↪ Protective mechanisms of the canal

A- The shutter mechanism:

- Contraction of the conjoint tendon during straining → straightens & descends downwards towards the inguinal ligament closing the posterior wall.

➤ Other mechanisms:

B- Obliquity of the canal:

- So, both rings aren't opposite to each other.

C- The extra-support of weak points of the canal:

- Behind the external ring (by conjoint tendon).
- In front of the internal ring (by condensation of fascia transversalis & fleshy fibers of internal oblique).

D- The cremasteric mechanism (Bulging mechanism):

- Contraction of cremasteric muscle during straining → bulge of spermatic cord in the middle $\frac{1}{3}$ of inguinal canal + elevation of the testis to close the external ring.

E- ↑ obliquity of the canal during straining:

- Fascia transversalis is attached to transversus abdominis muscle so during cough → contraction of muscle transversely and backward → displace fascia and ring laterally → increase of the obliquity of the canal (anti-hernia)

➤ Clinical points

↪ The inguinal ligament

- The lower free curved border of external oblique aponeurosis between pubic tubercle (medially) & A.S.I.S. (laterally).
- Its lower border is attached to fascia lata (deep fascia of thigh).
- Its free posterior margin fuses with lower end of fascia transversalis & fascia iliaca.
- In its medial ¼, the infolded part is thick & attached to ilio-pectineal line of pubic bone to form lacunar (**Gimbernat's**) ligament.

↪ Mid inguinal point

- Point mid way between ASIS & symphysis pubis.
- It is the surface anatomy of the external iliac artery.

↪ Midpoint of inguinal ligament

- Point mid way between ASIS & the pubic tubercle.
- ½ inch above it is the internal ring.

➔ **Why the hernia gives expansile impulse on cough?**

- Due to presence of sac which is continuous with peritoneum (not the content).

➔ **When hernia doesn't give expansile impulse on cough?**

- If strangulated.
- Fatty hernia of linea alba.
- A large sac (gives weak impulse).

➔ **Other sacs which gives expansile impulse on cough?**

1. Laryngocele (through thyrohyoid membrane).
2. Pneumatocele (suprapleural membrane or Simpson's fascia).
3. Empyema necessitans (chronic T.B. sinus).
4. Meningocele.
5. Saphena varix.
6. Large varicocele.

➔ **Fascia lata** causes arching of the inguinal ligament downwards.

Application: you must bend the legs during abdominal examination to relax the fascia lata → relax inguinal ligament → Relax the abdominal muscles.

➔ **OIH comes through the internal ring:**

- DIH comes from Hasselbach's triangle → internal ring test can differentiate between them.

➔ **OIH enters the canal from the internal ring:**

So the sac of the hernia is inside the canal & also inside the spermatic cord.

➔ **Testicular artery arises from the aorta.**

➔ **Cremasteric artery:** arises from inferior epigastric artery (branch of external iliac A).

➔ **Artery of the vas:** arises from inferior vesicular artery (branch of anterior division of internal iliac artery).

➔ **Ilioinguinal nerve:**

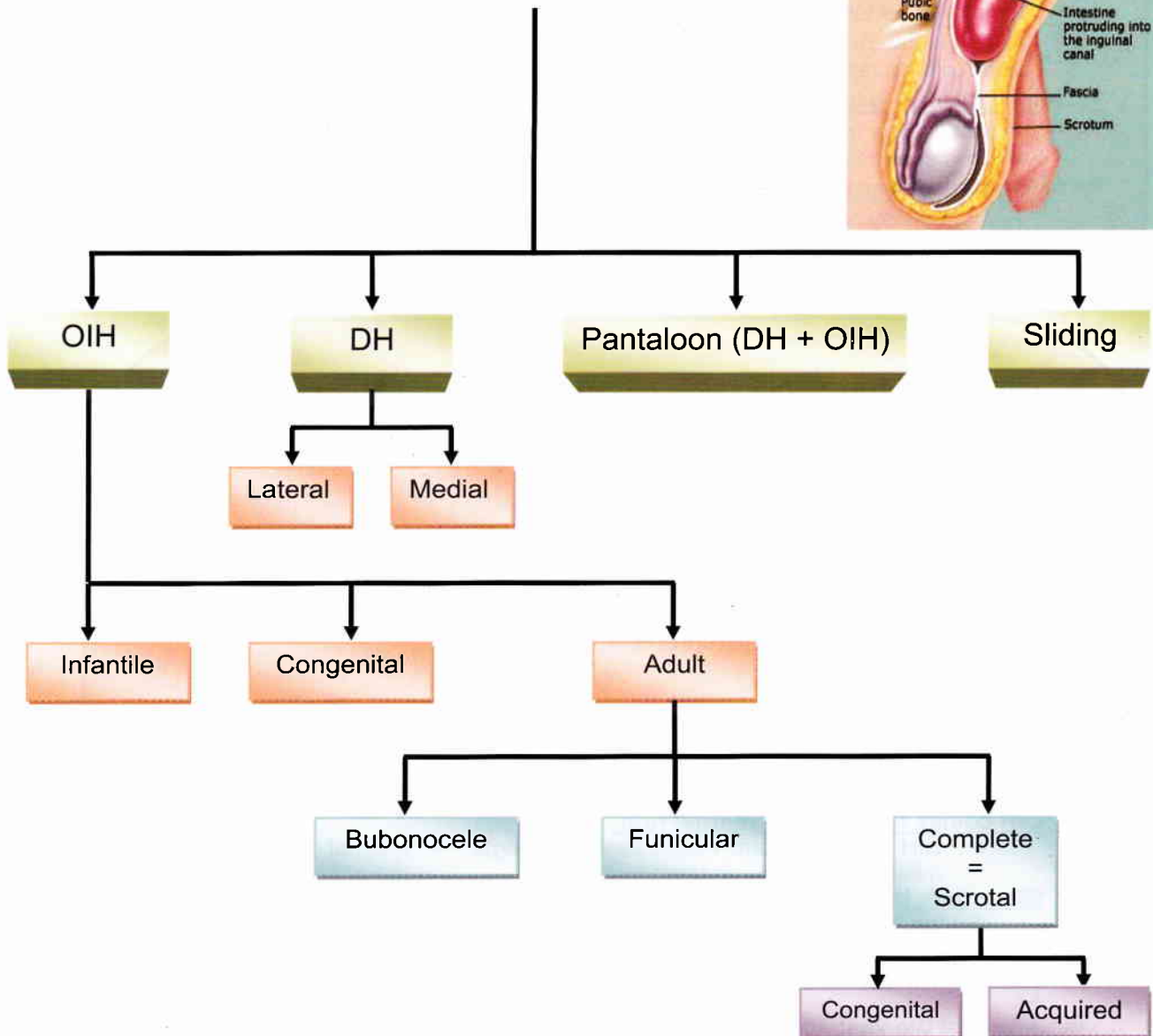
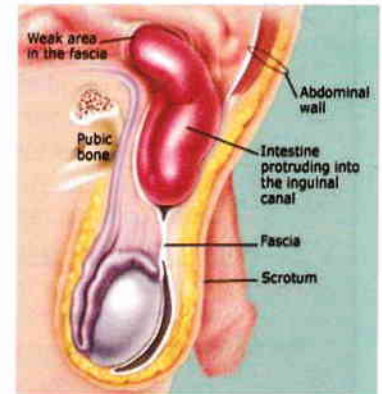
- Arises from lumbar plexus in psoas M.
- Supply conjoint tendon & sensory to the skin overlying the scrotum.

➔ **Genital branch of genitofemoral nerve:**

- Arises from lumbar plexus.

Types of Inguinal Hernia

1. O.I.H (Oblique inguinal hernia)
2. D.H (Direct hernia)
3. Pantaloon → both types DH, O.I.H **on the same side.**
4. Sliding inguinal hernia



Inguinal hernia in infants & children

- The hernia is always due to the presence of a preformed sac.
- It is always of oblique type.
- Operation can be performed at any age provided a skilled anesthetist is available.
- Herniotomy alone is performed. In infants the operation can be performed through the external ring without the need to open the inguinal canal.
- Recurrence is rare & is due to failure to ligate the sac at the proper neck.
- If strangulation occurs & neglected → testicular atrophy may occur.

Oblique Inguinal Hernia (O.I.H)

(Indirect inguinal hernia)

Incidence

- It is the commonest inguinal hernia (65%).
- Occurs at any age but more common in young age.
- Occurs in any sex but more common in males (20:1) due to weakness of the wall by the spermatic cord.
- 55% on the right side due to delayed descent of the right testis. However, it may be bilateral (12 %).

- Definition
- Incidence (male / right)
- Etiology (congenital / acquired)
- Pathology (covered / content/ defect / sac)
- Types
- Clinical Picture
- Treatment

Etiology

C. Congenital (Preformed) sac

- Due to **unobliterated** processus vaginalis or it **may be obliterated** by weak tissue → herniates later in life.

D. Acquired (Pulsion) sac

- Due to **weak** abdominal wall & ↑ intra-abdominal pressure.
- Weakness of fascia transversalis & widening of the internal ring.
- Occurs in adults.
- The testis is separated from the sac & lies behind it.
- Gradually enlarging inguinoscrotal swelling.

Pathology

Defect:

- Stretched deep inguinal ring **lateral to** inferior epigastric artery.

Sac:

- Present inside cord coverings **antero-lateral** to vas & vessels.

Contents:

- Small intestine, omentum or both (or other contents).

Coverings:

➡ In inguinal region

- 1- Internal spermatic fascia
- 2- Cremasteric muscle & fascia
- 3- *External oblique aponeurosis*
- 4- Camper's & Scarpa's fascia
- 5- Skin

➡ In the scrotum

- 1- Internal spermatic fascia.
- 2- Cremasteric muscle.
- 3- External spermatic fascia
- 4- Dartos muscle
- 5- Skin.

Classification

➡ According to Age of presentation:

- Congenital:** unobliterated process vaginalis. The hernia reaches down to the bottom of scrotum (Complete hernia). The testis lies among the content of the sac. Although called congenital it may appear in adult life.

- **Infantile:** operative finding. The tunica vaginalis extends upward to the external ring, so the sac passes down behind it. At operation the tunica is liable to be opened in mistake for the true sac (two sacs).
- **Adult type.**

➡ According to size:

a) **Bubonocele:**

- The hernia is limited to **inguinal canal** & is seen as a bulge or a swelling in the groin.
- The processus vaginalis is obliterated **at superficial ring**.

b) **Funicular hernia:**

- The hernia passes through external ring reaches down into scrotum reaching only the neck of scrotum.
- The processus vaginalis is obliterated only at its lower end just above the epididymis.
- The testis can be felt separate from hernia and below it.

c) **Complete (Scrotal) hernia: (the worst type)**

- Due to unobliterated processus vaginalis, may arise at adolescent.
- The hernia descends to the bottom of scrotum as a first presentation.
- It may be strangulated at the 1st time it appears.
- The testis is inside the sac.

Clinical Picture

Symptoms

- Clinical picture of swelling:
 - Reducible **swelling** (onset, course...etc.)
 - **Painless** (unless complicated).
- Clinical picture of complications.
- Clinical picture of precipitating factors.

Signs

General examination

- Search for other features of mesenchymal weakness
e.g. varicose veins, varicocele & flat foot.
- Signs of precipitating factor:
 - Chest → cough.
 - DRE → B.P.H.
- Signs of complications:
 - Inflammation.
 - Obstruction.
 - Strangulation.

Local examination

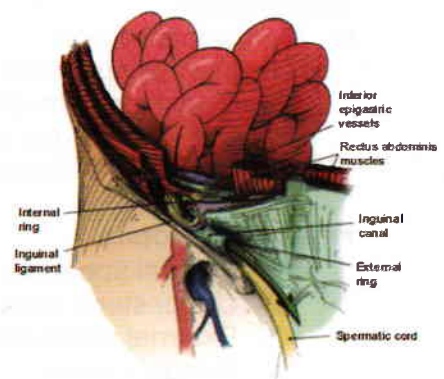
1. Inguinal or inguino-scrotal swelling.
2. Gives expansile impulse on cough.
3. Descends **downwards, forwards & medially**.
4. Reducible **upward, backwards & laterally**.
5. Internal ring test → **+ve**

Differential Diagnosis

1. Direct hernia
2. Dual hernia
3. Femoral hernia.
4. Varicocele
5. Congenital hydrocele



Right Oblique inguinal hernia



Direction of an indirect hernia

6. Infantile hydrocele of the cord.
7. Encysted hydrocele of the cord
8. Hydrocele of hernial sac.
9. Fibro-fatty lipoma of the cord

Complications See before.

Investigations See before.

Treatment of oblique inguinal hernia

I. Treatment of the precipitating factors to avoid recurrence

II. Surgical correction

1. Herniotomy:

- Done in congenital hernia (in children & infants).
- Opening of fundus of the sac → reduction of the contents → transfixation of the proper neck → excision of the sac.

2. Herniorrhaphy:

- Done in adults & elderly patients (as fascia transversalis is weak).
- Herniotomy + repair of posterior wall of inguinal canal.
- Disadvantages → tension – recurrence.
- Now, only indicated if infection or concomitant bowel resection where hernioplasty is contraindicated.
- Types of herniorrhaphy
 - i) **Marcy repair.**
 - ii) **Bassini repair.**
 - iii) **Shouldice repair.**

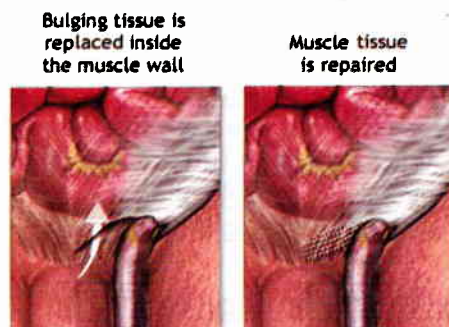
3. Hernioplasty

- Done esp. if there is:
 - i) Very weak abdominal wall.
 - ii) Large defect.
 - iii) Recurrent hernia.
- Types:
 1. **Darning**
Loose sutures for the gap between inguinal ligament & conjoint tendon with prolene sutures.
 2. **Grafting**
 - a) Natural by fascia lata or skin (**not done now**).
 - b) Synthetic (**the standard operation**).
 - Prolene mesh
 - Mersilene mesh
 - PTFE

4. TTT of associated sliding hernia, if present.



Intestine passes into the scrotum or groin



Hernioplasty using mesh

Direct Inguinal Hernia

Incidence

- Common in old aged male (in practice, females never develop direct hernia).
- It may arise in young age following appendectomy due to injury of ilioinguinal nerve supplying the conjoint tendon.
- 50% bilateral.

- Definition
- Incidence (male / bilateral)
- Etiology
- Pathology (defect / sac / content / coverings)
- Types (lateral / medial)
- Clinical Picture
- Treatment

Etiology

Always acquired

- Weakness of lower abdominal wall muscles & ↑ intra-abdominal pressure (chronic cough & BPH) usually associated with Malgaigne bulges.
- Defect in the conjoint tendon (funicular type).

Pathology

- **Defect:** Hasselbach's triangle medial to inferior epigastric artery or defect in conjoint tendon (according to type).
- **Sac:** present behind spermatic cord.
- **Contents:** Small intestine, omentum or both (or other contents).
- **Coverings:**
 1. Fascia transversalis.
 2. Stretched conjoint tendon.
 3. External oblique aponeurosis or external spermatic fascia.
 4. Superficial fascia.
 5. Skin.

Types

1. Lateral type:

- Bulges through **lateral part** of Hasselbach's triangle (fascia transversalis only).
- Characterized by **wide neck** → **less** liable for complications.
- **Never** descends to scrotum.

2. Medial type:

- Bulges through **medial part** of Hasselbach's triangle (conjoint tendon & fascia transversalis).
- Usually due to injury of ilioinguinal nerve → **paralysis of conjoint tendon**.
- Characterized by **narrow neck** → **more** liable for complications.
- **May** descends to scrotum (but never reaches its bottom).

Clinical Picture

Symptoms

- Clinical picture of swelling:
 - Reducible **swelling** (onset, course. etc.)
 - **Painless** (unless complicated),
- Clinical picture of complications.
- Clinical picture of precipitating factors.

Signs

➤ General examination

Search for other features of mesenchymal weakness e.g. varicose veins, varicocele & flat foot.

➤ **Local examination**

1. Inguinal swelling.
2. Gives expansile impulse on cough.
3. Descends **forwards**.
4. Reducible directly **backwards**.
5. Internal ring test → **- ve**.

Differential Diagnosis

1. Direct hernia.
2. Dual hernia.
3. Femoral hernia.
4. Varicocele.
5. Congenital hydrocele.
6. Infantile hydrocele of the cord.
7. Encysted hydrocele of the cord.
8. Hydrocele of hernial sac.

Treatment of Direct inguinal hernia

Treatment is essentially surgical

1. **Treatment of the Precipitating factors.**

e.g. Prostatectomy in SEP (before hernia repair).

2. **Surgery**

- Repair of the weak posterior wall of inguinal canal (Shouldice or Marcy repair is satisfactory).
- Mesh hernioplasty may be indicated.

N.B. Herniotomy is not usually needed (as sac is small & has an ill-defined neck).

3. **Truss** may be indicated if the patient is not fit for surgery

Causes of inoperability

- Bad general condition
- Recurrence
- Not liable for strangulation
- Very old age



Summary of differences between oblique & direct inguinal hernia

	Oblique inguinal hernia	Direct inguinal hernia
Incidence	More common	Less common
Age & sex	Any	Old males
Side	Unilateral or bilateral	Common bilateral (50%)
Size	Larger	Smaller
Shape	Oblong	Hemispherical
Descent	Downwards, forwards & medially	Forwards
Reducibility	Upwards, backwards & laterally	Backwards
Descent to scrotum	Sometimes	Rare
Complications	↑	↓
Internal ring test	+ve	-ve
External ring test!!!!	Impulse at tip of finger	Impulse at side of finger
Relation of neck of sac to inferior epigastric a. (intra-operative)	Lateral to the artery	Medial to the artery

MORE DETAILS

Bilateral direct inguinal hernia

- **Presented with** two bulges in the lower abdomen of a patient with weak abdominal muscles. This is called **Malgaigne bulges**.
- **Treatment options:**
 1. Repair using a giant prosthetic reinforcement of the visceral sac (**Stoppa repair**).
 2. Simultaneous laparoscopic repair is another appropriate method.
 3. Bilateral anterior repair through separate incisions can be done (traditionally, it has been associated with a recurrence rate approximately twice that of unilateral repair).

Dual hernia (Pantaloon) (Saddle bag)

- Both direct and indirect inguinal hernias arising at the same side with the inferior epigastric vessels between necks of the 2 sacs.

Sliding Inguinal Hernia (Hernia en glissade) (Organ forming part of the sac)

Incidence

- It occurs in patient with long standing hernia.
- The peritoneum of the viscus forms a part of the sac.
- Caecum, pelvic colon, bladder might slide.

Clinical picture

Sure diagnosis is intra-operative

- History of **long standing** hernia in obese elderly male.
- **Partially irreducible hernia**.
- **Double micturation** if the bladder is the sliding organ.
- Hernia is usually complete oblique inguinal hernia.

Treatment

- Freeing the sliding sac and the viscus wall from the surrounding structure → push them back behind fascia transversalis which is then repaired by multiple inverting sutures "**Bevan technique**" → then, hernioplasty.



sliding hernia :
organ forming part of the sac

Anatomy of Femoral Canal

Femoral sheath

- It surrounds the upper 4cm of femoral vessels (in the upper part of femoral triangle).

Site

Below inguinal (Poupart's) ligament in the upper part of thigh

- Ant. Wall:** fascia transversalis.
- Post. Wall:** fascia iliaca.

Size

- 4 cm long laterally & 1 cm medially.

Compartments

- Lateral compartment:** contains femoral a. & femoral branch of genitofemoral n.
- Middle compartment:** contains femoral vein.
- Medial compartment (femoral canal):** contains fat & LN of Cloquet.

Femoral Canal

- Femoral canal is the most medial compartment of femoral sheath.
- It is cone shaped & about 1.5 cm.

A. Inlet of the canal (Femoral Ring)

- It is the inlet of the femoral canal in abdomen.
- It is closed by a plug of fat called femoral septum.

Boundaries of femoral ring:

- Ant.** → Inguinal (**Poupart's**) ligament.
- Post.** → Pectineal (**Cooper's**) ligament.
- Lat.** → Femoral vein.
- Med.** → Sharp crescentic margin of lacunar (**Gimbernats**) ligament.

B. Outlet of femoral canal

- Saphenous opening & covered by cribriform fascia.
- Apex of the canal is closed thus, saphenous opening is found on the anterior wall of femoral sheath.

Contents

- Fat, lymphatic vessels & LNs of Cloquet.

Function

- Gives space for expansion of femoral vein with ↑ venous return during lower limb exercise.

Surgically important point

- The femoral hernia descends first downwards in the femoral canal, then forwards through the saphenous opening.
- Later if it becomes larger it curves upwards & (this is because of the attachment of Scarpa's fascia to fascia lata at a finger's breadth below the inguinal ligament).

- Abnormal obturator artery** may be present which represents **enlarged pubic branch of the inferior epigastric artery** when obturator artery is congenitally absent (30% of cases).
- In 10% of cases**, it's present in **dangerous site** (behind lacunar ligament, as we might cut the ligament during repair of hernia).
- In 20% of cases**, it's present in **safe site** behind the symphysis pubis or lateral to the ring.

Femoral Hernia

Definition

- ⇒ **Pathological:** protrusion of a viscus or a part of a viscus usually within peritoneal sac through the femoral ring into the femoral canal.
- ⇒ **Clinical:**
- 1- Reducible or gives history of reducibility
 - 2- Gives expansile impulse on cough.
 - 3- On the anatomical site of hernia (femoral region).

- Definition
- Incidence
- Etiology
- Pathology
- Clinical Picture
- Investigations
- Treatment

Incidence

- Higher incidence on the Rt. side (2:1).
- It may be bilateral in 20% of cases.
- 3rd most common hernia after inguinal and incisional hernias, representing 20% of female hernias and 5% of male hernias.
- More common in females (2:1), middle-aged (20-40 years) due to:
 1. Repeated pregnancy → ↑ intra-abdominal pressure + ↑ progesterone
 2. Wide pelvis → wide femoral ring.
 3. Small sized femoral vein → wide femoral ring.
 4. Vasodilatation & vasoconstriction of femoral vessels → weakness of fat.
 5. Less developed ilio-psoas muscles.

■ The inguinal hernia remains the commonest among any age or sex.

Etiology

A- Congenital (rare):

- 1- Laugier's femoral hernia: through Gimbert's ligament (does not reach the femoral canal). ☐
- 2- Cloquet's (pectineal) femoral hernia: under sheath of pectineus muscle.
- 3- Narath's type of femoral hernia occurs with congenital hip dislocation.

B- Acquired (it is almost always acquired):

- 1- Raised intra-abdominal pressure (precipitating factors) due to:
 - Chronic cough.
 - Obesity.
 - Straining (e.g. constipation, prostatism).
 - Abdominal swelling (splenomegaly).
- 2- Weak anterior abdominal wall due to:
 - Repeated pregnancy.
 - Senility.

Pathology

➤ Defect:

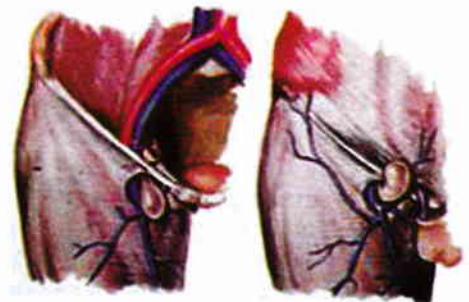
- Through the femoral ring.

➤ Sac:

- The sac passes downwards in the femoral canal then forward stretching the cribriform fascia (of saphenous opening) then upwards & laterally.
- The neck of the sac is narrow making femoral hernia more liable to complications (irreducibility & strangulation).

➤ Contents:

- Usually omentum, bowel or part of circumference of bowel (**Richter's hernia**) which is very common.



➤ Coverings:

1. Femoral sheath (when small).
2. Stretched cribriform fascia (when it comes outside saphenous opening).
3. Superficial fascia (Scarpa's fascia).
4. Subcutaneous fat.
5. Skin.

Clinical Picture

A- Clinical Picture of the case: *rounded painless swelling (unless complicated), characterized by:*

- 1- Reducible (downwards, backwards then upwards) or gives history of reducibility.
- 2- Gives expansile impulse on cough (unless complicated).
- 3- On the anatomical site of hernia (below the medial part of inguinal ligament, 4 cm below and lateral to pubic tubercle).

B- Clinical picture of complications (if present)

The most liable for strangulation

C- History of precipitating factors: e.g. obesity or repeated pregnancy

➤ **Femoral hernia may present for the 1st time with strangulation (40%) especially Richter's:**

- Acute painful groin swelling ± features of intestinal obstruction.
- Hernia is irreducible with no impulse on cough, tender and tense.

Complications *See before*

▪ The most common is:

- 1- Irreducibility.
- 2- Strangulation.

DD

(Swelling in femoral triangle)

Reducible	Irreducible
- Reducible inguinal hernia.	- Irreducible inguinal hernia.
- Saphena Varix	- Subcutaneous lipoma.
- Femoral a. aneurysm.	- Inguinal lymphadenopathy (L.N. of Cloquet)
- Psoas abscess	- Mal-descended testis.

DD of Strangulated femoral hernia

1. Acute inguinal lymphadenitis.
2. Sub-inguinal abscess (FAHM, tenderness + no abdominal symptoms).
3. Torsion in maldescended testis.
4. Rupture adductor longus tendon (history of trauma or sudden strain, ecchymosis, tenderness + no abdominal symptoms).
5. Anterior dislocation of the hip joint.

Investigations

▪ As before.

Treatment

Surgery is the only line of treatment (Truss is contraindicated)

A- Prophylaxis: avoid predisposing factors.

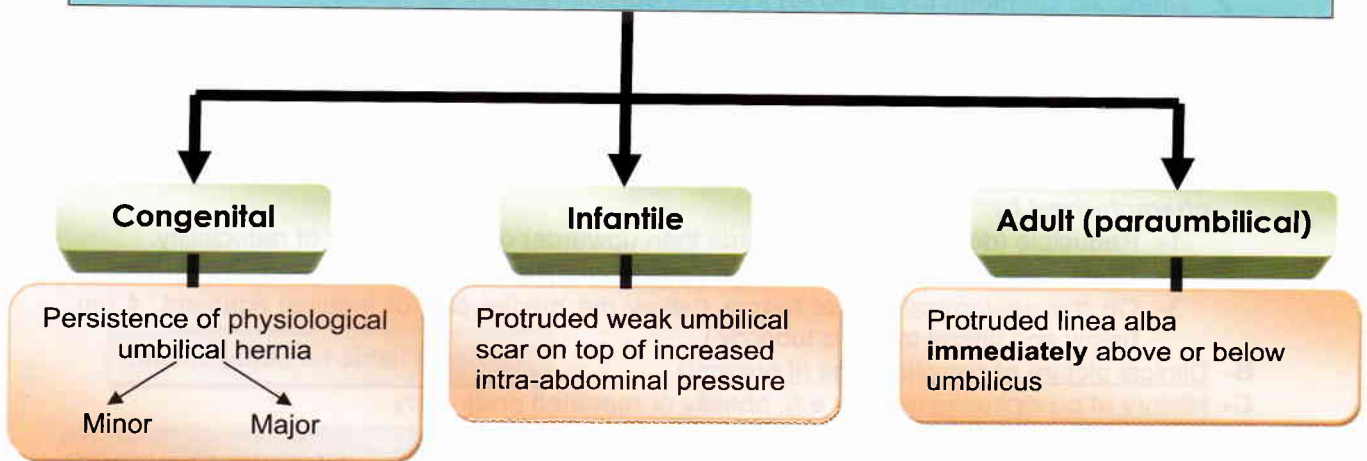
B- Treatment of precipitating factors

C- Curative: depends on approximation of the pectineal ligament to the ilio-pubic tract via:

- 1- Low approach (Lockwood's).
- 2- Inguinal approach (Lotheissen's).
- 3- Pre-peritoneal (McEvedy's): suitable for uncomplicated or complicated cases.
- 4- Laparoscopic approach.

D- Palliative: Truss is contraindicated due to high possibility of strangulation.

Umbilical Hernia



Congenital umbilical hernia (Exomphalos = Omphalocele)

Definition

⇒ **Pathological:** protrusion of a viscus or part of the viscus through congenital defect in abdominal wall.

⇒ **Clinical:**

1. Reducible or gives history of reducibility
2. Gives expansile impulse on cough.
3. On the anatomical site of hernia.

⇒ **It presents at birth.**

Etiology

- Failure of all or part of the midgut to return to the abdomen (persistence of physiological umbilical hernia)
- There is a defect in the anterior abdominal wall.

- Definition
- Etiology
- **Types (minor-major)**
- Pathology
- Complications
- Treatment



Intestine protruding through abdominal wall defect

Types



Exomphalos minor



Exomphalos major

I. Exomphalos minor

- **Defect:** small (< 5 cm) at the umbilicus (central cord).
- **Sac:** small (peritoneum).
- **Coverings:** Wharton's jelly and layer of amniotic membrane.
- **Content:** one loop of small intestine or Meckel's diverticulum.
- **Complications:** during ligation of the umbilical cord, a loop of intestine may entangled in the ligature accidentally → resection (umbilico-enteric fistula?).
- **Treatment:**
 - Content is reduced.
 - Sac is excised.
 - Defect is repaired in layers.

II. Exomphalos major

- **Defect:** larger (> 5 cm) in the center of abdominal wall (usually supra-umbilical, eccentric cord).
- **Sac:** large (peritoneum).
- **Coverings:** only a layer of amniotic membrane covers the sac.
- **Content:** any abdominal viscera e.g. **liver** (specifically, it is adherent to the sac), stomach, bowel, spleen...etc.
- **Complications:** rupture of the sac and coverings may occur → infection → peritonitis (the cause of death).
- **Treatment:** **Urgent surgery**
 - A. If the abdominal cavity can accommodate the contents:**
Primary closure under moderate tension is done.
 - B. If the abdominal cavity can't accommodate the contents:**
 - 1. Skin flap closure:**
 - Undermine the skin and close as one layer by with simple sutures.
 - Two release incisions may be needed.
 - If survives → definitive repair after several months (*Usually fails*).
 - 2. Recently → staged closure:**
 - Cover the defect with synthetic material
 - Apply gradual pressure to ↑ size of abdominal cavity.
 - If survives → definitive repair.

N.B. If associated with multiple congenital anomalies (or unfit for surgery) → paint the viscera with alcohol and conserve.

Gastroschisis:

- It is a congenital umbilical hernia but the peritoneal sac is not intact.
- The viscera may or may not be covered with amniotic membrane.
- There is controversy whether to consider it a separate entity or not.
- Management of gastroschisis remains the same as congenital umbilical hernia.

Infantile Umbilical Hernia

Definition

- ⇒ **Pathological:** protrusion of a viscus or part of the viscus through a defect in abdominal wall.
- ⇒ **Clinical:**
 1. Reducible or gives history of reducibility.
 2. Gives expansile impulse on cough.
 3. On the anatomical site of hernia.
- ⇒ **It presents few weeks or months after birth.**

- Definition
- Incidence
- Etiology
- Pathology
- Clinical Picture
- Complications
- DD
- Treatment

Incidence

- ⇒ More in black races.

Etiology

- ⇒ Weak umbilical scar (due to infected umbilical stump, Cicatrix).

Pathology

- **Defect** is **exactly at** the umbilicus.
- **Sac:** peritoneum, small, conical with wide neck.
- **Contents:** omentum, bowel or both.
- **Coverings:** stretched umbilical scar & extra-peritoneal fat.
- **Neck** of the sac is wide → rare to be strangulated.



Protruded umbilical Scar

Clinical picture

- A. Clinical Picture of the case: **painless swelling (unless complicated), characterized by:**
 1. Reducible or gives history of reducibility.
 2. Gives expansile impulse on cough or crying (unless complicated).
 3. On the anatomical site of hernia.
- B. Clinical picture of complications (if present).
- C. History of precipitating factors e.g. phimosis (constricted foramen of prepuce).

Fate

- Spontaneous closure in the 1st few months of life is the usual fate (85%).

Complications

- Rare.

DD

	Congenital umbilical hernia	Infantile umbilical hernia
Covering	Amniotic membrane	Skin
Presentation	Dated since birth	After few weeks (falling of umbilical stump) → umbilicus is everted.

Treatment

Reassurance of the parents & **follow up** are the usual measures.

(The defect usually **closes spontaneously** within 2 years in 95% of cases)

- **Treatment of the precipitating factors** e.g. phimosis → circumcision.
- **Coin & plaster strap!!!** (No need as defect closes spontaneously).
- **Indications for surgical correction:**
 1. Large defect (> 2 fingers).
 2. > 2 years old.
 3. Strangulation.

→ Anatomical repair with proline sutures.

Adult Umbilical Hernia (Para-umbilical)

Definition

- **Pathological:** protrusion of a viscous or part of the viscous usually within peritoneal sac through a defect in linea alba immediately above (more common) or below the umbilicus.
- **Clinical:**
 - Reducible or gives history of reducibility
 - Gives expansile impulse on cough.
 - On the anatomical site of hernia.

- Definition
- Incidence (**middle-aged females**)
- Etiology
- Pathology
- Clinical Picture (READ + **intertrigo**)
- Complications
- Investigations
- Treatment

Incidence

- More common in middle-aged females due to multiple pregnancies.

Etiology

1. Raised intra-abdominal pressure (precipitating factors) due to:
 - Chronic cough.
 - Obesity.
2. Weak anterior abdominal wall due to: repeated pregnancy.

Pathology

- **Defect:**
 - Defect in the linea alba just above (more common because linea alba is wide and weak from stretch by the stomach) or below (less common) the umbilicus.
 - It does not occur in the side of the umbilicus because of the rectus abdominis muscle.
 - Linea alba doesn't perforate, but becomes weak & lax, so peritoneum bulges in between the weak points (i.e. it is multilobulated).
- **Sac:**
 - Has a narrow neck.
 - Multi-loculated with adhesions inside the sac which are very common (especially at the fundus) rendering the hernia irreducible.
- **Contents:**
 - It might contain omentum, **transverse colon** or small intestine (so, it is liable for strangulation & becomes gangrenous).
- **Coverings:**
 - Skin, subcutaneous tissue & extra-peritoneal fat.

Clinical Picture

- A. Clinical picture of the case:** **painless swelling (unless complicated), characterized by:**
 1. Reducible (directly backwards) or gives history of reducibility.
 2. Gives expansile impulse on cough (unless complicated).
 3. On the anatomical site of hernia.
 4. The hernia may **be large** to the size of an orange but the neck of the sac remains dangerously small.
 5. The overlying skin may be affected by **intertrigo** → should be treated first as it may cause post-operative wound sepsis & recurrent hernia.
- B. Clinical picture of complications (if present).**
- C. History of precipitating factors** e.g. obesity of repeated pregnancy.



Umbilical hernia



Umbilical hernia during cough

Complications

- As before.
- The commonest is irreducibility as the sac of paraumbilical hernia is multi-loculated (as linea alba is sieve-like) and due to presence of omentum.
- Hydrocele of the hernial sac does not occur in this hernia as the hernia is not in a dependant position (occurs only with ascites).

Investigations

- See before.

Treatment

Surgery is the only method of treatment

A- Prophylaxis: avoid predisposing factors.

B- Curative:

- Treatment of predisposing factors first to avoid recurrence & TTT of intertrigo if present.
- Then surgical repair is done:
 - 1- Anatomical repair (repair of the whole linea alba).
 - 2- Mayo's repair (repair the defect only).
- If the hernia is large → post-operative ventilator is advised (because reduction of a large hernia will cause respiratory distress since the abdominal cavity needs time to accommodate the content).

C- Palliative: truss is contraindicated due to high possibility of strangulation.

Epigastric Hernia

(Fatty Hernia of Linea Alba)

Definition

- Protrusion of the extra-peritoneal fat through a defect in the supra-umbilical part of linea alba and is called "**fatty hernia of linea alba**". It is irreducible, giving no impulse on cough sometimes painful due to incarceration of fat.
- As the protrusion enlarges, the fat pulls through the defect small peritoneal pouches which contain intestine or omentum and is called "**epigastric hernia**".

- Definition
- Pathology
- Clinical Picture
- Complications
- Investigations
- Treatment

- Greater omentum
- Interval dyspepsia
- Early, fatty linea alba → acquire omentum → epigastric hernia

Components

- **Sac:** If present, it is usually small and has narrow neck.
- **Contents:** Usually a piece of **greater omentum**.
- **Coverings:** Subcutaneous tissue and skin.

Clinical Picture

Most of the cases are asymptomatic

- On examination: **painless swelling (unless complicated), characterized by:**
 - 1- Reducible or gives history of irreducibility.
 - 2- Gives expansile impulse on cough (usually absent early as it contains fat, fatty hernia of linea alba).
 - 3- On the anatomical site of hernia "**separated from the umbilicus by interval**".
- It might contains part of the greater omentum (decreases the mobility of the stomach) and gives dyspeptic symptoms resembling peptic ulcer but here there is epigastric swelling.

Complications

- As before.

Investigations

- As before.

Treatment

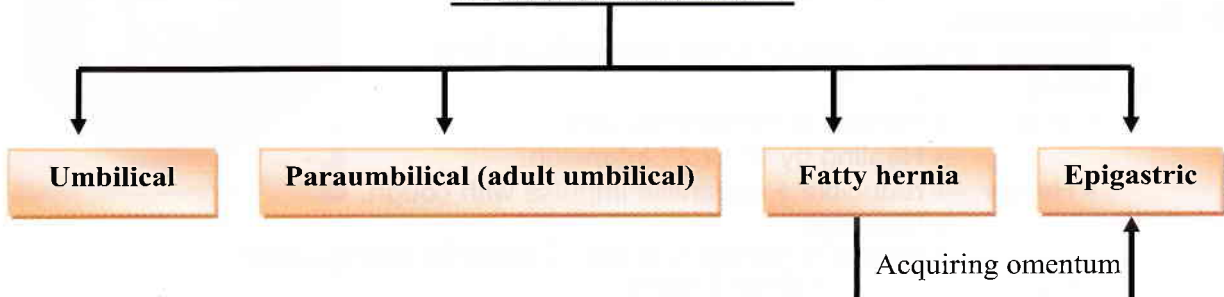
A- Prophylaxis: avoid predisposing factors.

B- Curative:

- Treatment of predisposing factors first to avoid recurrence.
- Then, surgical repair is done:
 - 1- Mayo's repair.
 - 2- Anatomical vertical repair of the linea alba.
 - 3- Mesh repair if large to avoid recurrence.



Hernia of Linea Alba



Incisional Hernia

(Acquired hernia) (Man-made hernia) (Ventral hernia)

- It is a hernia that develops at the site of a previous abdominal incision.
- It develops immediately or early in the post-operative period.

Etiology

Pre-operative

1. Weak abdominal muscles.
2. Obesity.
3. Chronic cough
4. Chronic constipation
5. Senile enlarged prostate.
6. Nature of 1^{ry} disease e.g. peritonitis, neglected I.O. or abdominal malignancy.
7. General debilitating disease e.g. uremia, obstructive jaundice or DM.

Operative

1. Excessive trauma to the tissues.
2. Bad hemostasis, with loss of > 1000 ml blood during operation.
3. The repair is too loose or too tight.
4. Vertical more than transverse incisions.
5. Muscle cutting more than muscle splitting incisions.
6. Closure of abdominal wall with absorbable sutures. It's recommended to take good bites on either sides of the wound using non-absorbable sutures as prolene.
7. Insertion of foreign bodies like tube drains in the main wound.

Post operative

1. Wound infection (→ friable tissues, consumption of nutrients, dissolve of catgut).
2. Vomiting or vigorous coughing due to bad recovery from anesthesia.
3. Early return to work.
4. Persistent precipitating factors.
5. Abdominal distension due to prolonged paralytic ileus.
6. Wound hematoma.

Types

- a. Defective Type (cicatrical).
- b. Paralytic type.

Clinical picture

A- History of :

1. The cause.
2. Type of operation (especially midline & subcostal incisions), timing, postoperative period (vomiting, early ambulation, wound infection).

B- On examination:

☞ **General:** anemia, obesity, chest problems or BPH.

☞ **Local:**

- Scar → Vertical or transverse, ugly.
→ Healing by 1^{ry} or 2^{ry} intention.
- Hernia → reducible + expansile impulse with cough.
→ Intertrigo.
→ Defect: • Narrow & sharp → liable for strangulation
• Wide & blunt



- Abdomen → Divarication of recti.
→ HSM & ascites.

C- Clinical picture of complications if present.

Complications

- As before

Investigations

- As before

Treatment

Prophylactic treatment

1. Avoid the precipitating factors e.g. reduction of weight, treatment of anemia.
2. Use of non-absorbable prolene sutures in closure of the abdominal incision.

Curative

- **Treatment of predisposing factors first to avoid recurrence.**
- Then surgical repair is done:
 1. Anatomical repair.
 2. Maingot (Keel) repair.
 3. Cattell's repair.
 4. Tension-free hernioplasty especially if large.

Palliative (abdominal corset)

- If the operation is contra-indicated.

The hernia might attain large size because:

1. It is painless.
2. The patient has just undergone an operation.
3. Psychologically afraid of operations.

The hernia might be so big & in these circumstances

- i) **Skin preparation will be important (treatment of intertrigo).**
- ii) Post-operative ventilator might be needed. (Sudden reduction of hernia followed by its closure → ↑ intra-abdominal pressure → interferes with diaphragmatic mobility → respiratory distress).
- iii) Recently → gradual pneumo-peritoneum, introduction of gradually ↑↑ pressure of air inside peritoneum → to adapt high pressure before reduction.

Recurrent Hernia

- It might occur after repair of any type of hernia.
- Its incidence is about 10 - 15%.
- Usually within 1 year from the operation.

Etiology

As incisional hernia + incomplete removal of the sac + missed sac (as dual hernia) + displacement of prosthesis + use of prosthesis of inadequate size.

Treatment

- Correction of predisposing factors + hernioplasty through a different approach to avoid dissection through the scar tissue.

Burst Abdomen

Definition

- Complete disruption of an abdominal incision in the early post-operative period.

- ▶ **Etiology:** sepsis, sutures, serious dis.
- ▶ **C/P:** red sign ± I.O.
- ▶ **Invest.:** culture, imaging
- ▶ **TTT:** prophylactic + active (tension sutures)

Etiology

Etiology of incisional & recurrent hernia is the same as burst abdomen

⇒ **As incisional hernia.**

⇒ **Abdomen is likely to burst if:**

- It is swollen for any reason such as ileus, intestinal obstruction or large tumor.
- Severe intra-abdominal sepsis.
- Suturing the abdomen with absorbable sutures.
- Suturing in layers taking bites of tissues that are too small.
- Debilitating disease, e.g. uremia, obstructive jaundice or malignancies.

⇒ **Abdomen will never burst if:**

- Non-absorbable sutures.
- Taking wide bites of tissues.
- You use delayed skin suture if the wound is infected or potentially so.

Types

- **Partial:** deep layers burst but the skin is intact producing → incisional hernia.
- **Complete:**
 - a) If the intestine prolapsed out of the wound → called evisceration.
 - b) If the intestine is retained inside abdomen → called wound dehiscence.

Clinical picture

- Usually occurs at 6th - 8th day post-operative.
- A serosanguinous discharge is often a warning sign and called → **Red sign** (most important)
- The patient feels as if something gives away.
- Symptoms of intestinal obstruction may be present.

Investigations

⇒ **Lab:**

- Wound and tissue culture.
- Blood tests.

⇒ **Imaging:**

- X-ray to evaluate the extent of the wound separation.
- U/S for pus pockets.
- CT for pus pockets.

Treatment

➤ **Prophylaxis:** Avoid and treat any predisposing factor.

➤ **Active TTT**

⇒ **Complete burst**

- **Pre-operative measures:**
 1. Cover the wound with a sterile towel and warm saline.
 2. Ryle's tube + IV fluids + antibiotics.

- Operative:
 1. Protruded intestinal loop are washed with saline and returned to the abdomen, the omentum is spread over the intestine, the abdominal wall is closed as one layer using **tension “through and through” suture**.
 2. Abdominal binder is used post-operative.

Oral Questions

➤ What are the Diseases of the umbilicus?

1. Umbilical fistula:

- ❑ **Congenital:** patent vitello-intestinal duct.
- ❑ **Traumatic.**
- ❑ **Inflammatory:** from T.B. of small intestine
- ❑ **Malignant:** carcinoma of transverse colon ulcerating.
- ❑ **Urinary fistula:** may be congenital or acquired (rarely).
- ❑ **Biliary fistula:** very rare due to operative bile duct injury during cholecystectomy.

2. Umbilical sinus:

- ❑ Discharge pus & is due to either an abdominal wall abscess or umbilical infection.
- ❑ Pilonidal sinus of the umbilicus is rare, but leads to persistent discharge.

3. Umbilical stone:

- ❑ Due to chronic inflammation of umbilicus & form umbilical granuloma.
- ❑ Stone should be removed. The granuloma is excised by diathermy & antiseptics applied.

4. Umbilical polyps:

- ❑ Due to presence of the umbilical extremity of VID, whenever everted outwards.
- ❑ The epithelial surface undergoes irritative hyperplasia from friction leading to formation of a polypoid mass at the bottom of the umbilicus. It should be excised.

5. Umbilical granuloma:

- ❑ A mass of granular tissue due to chronic infection of umbilical scar.
- ❑ **Treatment:** curetted or cauterized.

6. Umbilical hernia

7. Umbilical tumors:

- ❑ **Squamous cell carcinoma:** is rare. It gives metastasis to axillary & inguinal L.N.s on both sides.
- ❑ **2ry carcinoma nodules:** may be present at the umbilicus due to spread from carcinoma of stomach, pancreas, liver or breast.

➤ Complications of abdominal incisions

- 1. Hematoma:** this may be due to a bleeding tendency or more commonly due to careless surgical haemostasis. If the hematoma is small it can be left for spontaneous absorption but if enlarged or of a large size it should be evacuated to avoid secondary infection.

- 2. Infection.**

3. **Wound disruption** (burst abdomen): this is a serious complication which may be due to an incisional hernia or may even cause mortality.
4. **Incisional hernia.**
5. **Desmoid tumor.**

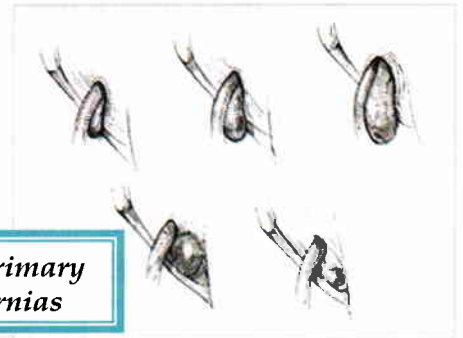
Classifications of Inguinal Hernias

1. (Nyhus) classification:

- Type 1 Indirect hernia with normal internal ring.
- Type 2 Indirect hernia with dilated internal ring. Posterior wall is intact.
- Type 3 Posterior wall defect.
- Type 4 Recurrent hernia.
 - A Direct inguinal hernia.
 - B Indirect inguinal hernia. Internal ring dilated, posterior wall defective.
 - C Femoral hernia.

2. Gilbert classification:

- ☞ It is based on evaluating 3 factors:
- 1) Presence or absence of a peritoneal sac.
 - 2) Size of the internal ring.
 - 3) Integrity of the posterior wall of the canal.



Gilbert classification for primary and recurrent inguinal hernias

Type	1	2	3	4	5
Internal ring	<1 cm	<=2 cm	>2 cm	Normal	Normal
Peritoneal sac	Y	Y	Y	N	N
Posterior wall	Intact	Intact	Not Intact	Not Intact	Not Intact

Rare Types of External Hernia

Lumbar Hernia

⇒ Anatomy of the lumbar triangles:

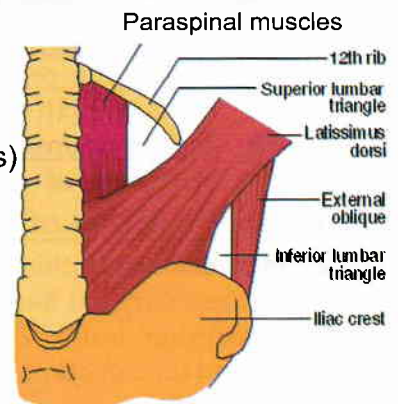
1. Superior lumbar triangle (Grynfeltt triangle):

- Boundaries:
 - Superiorly: last rib.
 - Medial: para-spinal muscles (sacrospinalis)
 - Lateral: latissimus dorsi & internal oblique

2. Inferior lumbar triangle (Petit triangle):

- Boundaries:
 - Medial: latissimus dorsi.
 - Lateral: external oblique.
 - Base: iliac crest.

Surgical importance: lumbar hernia



⇒ **Lumbar hernia:**

- Lumbar hernia presents in the superior or inferior lumbar triangle.
- Most hernias are large & reducible, with impulse on cough & have a wide neck.
- It is usually congenital or associated with muscle weakness due to previous operation (related to scar of previous operation).
- **DD** : Lipoma.
- **Treatment** by abdominal corset or surgery.

Phantom Hernia

- Local muscular paralysis as in poliomyelitis.

Obturator Hernia

- **More common in females**☑
- Through the obturator foramen (compresses the obturator nerve pain in the medial side of the thigh).
- **Pain and tenderness over medial side of thigh ↑** on doing extension and adduction or medial rotation of the hip (**Howship sign**) and if complicated will be referred to the knee☑).
- Absent adductor reflex in the thigh (**Hannington-Kiff sign**) may be positive.
- **Diagnosed** by CT.
- **Treatment:**
 - A posterior approach, either open or laparoscopic, is preferred to provide direct access to the hernia.
 - The obturator foramen is repaired with sutures or a small piece of prosthetic mesh, with care to avoid injury to the obturator nerve and vessels.

Gluteal and Sciatic Hernia

Origin

- Through the greater and lesser sciatic foramen respectively.

Clinical picture

- Sciatica (compression on sciatic nerve).
- Uncomfortable or slowly enlarging mass in the gluteal region.
- Intestinal obstruction.

Investigations

CT scan.

Treatment

- **Trans-peritoneal approach:** is preferred if bowel obstruction or strangulation is suspected.
- **Trans-gluteal approach:** in case of sure diagnosis and reducible hernia.

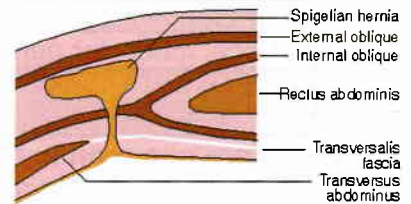
Perineal Hernia

- **This type of hernia is very rare and includes:**
 - Post-operative hernia through a perineal scar which may occur after excision of the rectum.
 - Medial sliding perineal hernia, which is a complete prolapse of the rectum.
 - Antero-lateral perineal hernia, which occurs in women and presents as swelling of the labium majus.
 - Postero-lateral perineal hernia, which passes through the levator ani to enter the ischio-rectal fossa.

- **Symptoms**
 - Worsened by sitting or standing.
 - Bulge (frequently detected on bimanual rectal-vaginal examination).
- **Treatment:** repaired through trans-abdominal approach or combined trans-abdominal and perineal approach + mesh.

Spigelian Hernia

- It usually occurs in old **female**.
- It is due to defect in the semi lunar line at the level of arcuate line.
- Early, it contains fat, later on, a full peritoneal sac develops.
- Patients often present with localized pain in the area without a bulge because the hernia lies **beneath the intact external oblique aponeurosis**.
- **US or CT** to establish the diagnosis.
- It's liable to strangulation with its relatively narrow neck. So anatomical repair is the treatment of choice.
- Recurrence is uncommon.



A Spigelian hernia emerging lateral to the rectus sheath.

Interstitial Inguinal Hernia

- Hernia in between layers of abdominal wall.
- Commonly in male.
- It may associate with inguinal or femoral hernia.
- It may be inguino-superficial, intra-muscular or pre-peritoneal.

Divarication of Recti Abdominis "Diastasis Recti"

- **Definition:** diffuse widening and attenuation of the linea alba without a fascial defect.
- Seen in elderly multiparous females.
- **On examination:**
 - Fusiform linear bulge between the two rectus abdominis muscles without a discrete fascial defect.
 - When the patient strains, a gap can be seen between the recti through which the abdominal contents bulge.
 - When the abdomen is relaxed the fingers can be introduced between the recti.
- **Treatment:** anatomical repair or mesh repair.

Amyand's hernia

- Rare form of inguinal hernia, appendix is included and becomes incarcerated.
- Treated by combined appendectomy and hernia repair.

Internal Hernia

Definition

- Part of small intestine entrapped one of the retroperitoneal fossa or congenital mesenteric defect.

Sites

- 1- Congenital/acquired diaphragmatic hernia.
- 2- Fossa of Winslow.
- 3- Duodenal retroperitoneal fossa.
- 4- Caecum/ appendicular retroperitoneal fossa.
- 5- Hole in transverse mesocolon.
- 6- Hole in mesentery.
- 7- Hole in broad ligament.

1. Internal (e.g. Hiatus hernia).
2. External.

a) Common:

- 1- Inguinal region (1st most common, may be direct or indirect)
- 2- Incisional (post-operative hernia) → 2nd most common.
- 3- Epigastric region.
- 4- Femoral region.
- 5- Umbilical region: may be umbilical or paraumbilical hernia.

b) Rare types.

Anatomy of common sites of internal hernia

The Diaphragm

- Origin:**
 - **Sternal:** back of xiphoid process.
 - **Costal:** lower 6 costal cartilages.
 - **Vertebral:** 2 crura; Rt. crus from front part of upper 3 lumbar vertebrae, Lt. crus from front of upper 2 lumbar vertebrae, 5 arcuate ligaments.
- Insertion:** central tendon.
- Surgical importance:**
 - ⇒ Openings of the diaphragm:
 - 1- **3 Large opening** (hiatus hernia through esophageal hiatus).
 - 2- **Foramen of Morgagni or Magendi:** between sternal and costal origins of the diaphragm through which para-sternal hernia may develop.
 - 3- **Foramen of Bochdalek:** due to persistent pleura-peritoneal canal (triangular gap between the last rib and diaphragm), through which posterior hernia develops.

Fossa of Winslow (Opening of lesser sac)

- Boundaries:**
 - **Superior:** caudate process of liver.
 - **Inferior:** 1st part of duodenum.
 - **Posterior:** IVC.
 - **Anterior:** **Free border of lesser omentum containing:**
 - Portal vein.
 - CBD anterior and to the Rt.
 - Hepatic artery: anterior and to the left.
- Surgical importance:**
 1. Site of internal hernia.
 2. Porto-caval anastomosis.
 3. Pringle's maneuver if bleeding occurs during cholecystectomy.
 4. Site of exposure of supra-duodenal portion of CBD for stone removal.

Mesentery

- The small gut is suspended by its mesentery which extends from the Lt. side of the 2nd lumbar vertebrae to the Rt. Iliac fossa crossing the 3rd part of duodenum, aorta, IVC and Rt. ureter.
- Ascending and descending colons are covered on the front and side by peritoneum but not from behind (i.e. they are retroperitoneal).
- Transverse colon has a transverse mesocolon, which is formed of 2 layers attached to the anterior border of pancreas.
- Sigmoid colon has sigmoid mesocolon, which has 2 limbs, meeting each other to form inverted V-shaped mesentery, with its apex directed up.
- The Lt. ureter descends behind apex.
- Superior rectal veins run along Rt. Limb.

Rectus sheath hematoma

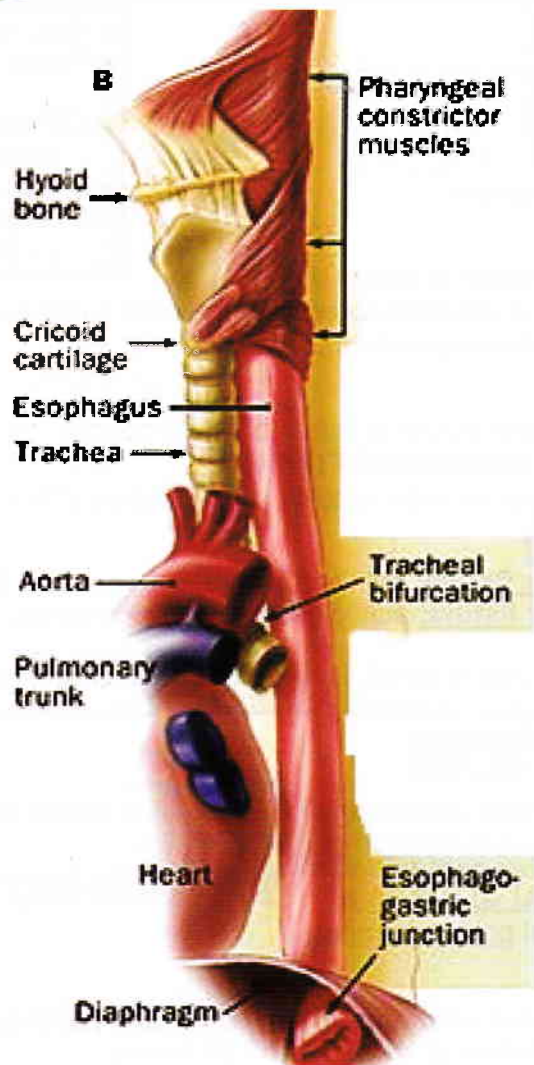
Etiology

- Abdominal wall trauma blunt or iatrogenic (careless surgical hemostasis)
- Following abdominal (open or laparoscopic) operations injuring the superior or inferior epigastric vessels → painful tender swelling over the rectus muscle with overlying skin ecchymosis.

Treatment

- Surgical evacuation and ligation of the injured vessel.

CHAPTER 2



THE ESOPHAGUS

Congenital Diseases of the Esophagus

Congenital Esophageal Atresia

Definition

- Failure of recanalization of foregut.

Incidence

- 1: 4,000.
- Male > female.

Etiology

- The condition is due to defect in division of proximal foregut into ventral tracheo-bronchial tube & dorsal esophageal tube.

Types

1. Blind upper pouch & fistula of lower pouch
→ **COMMONEST (87%)**
2. Rare types include: atresia without fistula (7%)

Pathology

- a. **In upper fistula:** milk and saliva → trachea → bronchopneumonia.
- b. **In lower fistula:** acidic gastric juice → trachea → acid pneumonia (fatal)

▶ Aspiration of all types of fistula due to overflow of saliva

Clinical Picture

- ▶ Any newborn presenting with frothy saliva should be considered as having esophageal atresia until proved otherwise.

1. Antenatal diagnosis: (By obstetrician)

- Maternal polyhydramnios.

2. At birth:

- Continuous pouring of saliva since birth (**pathognomonic**).
- Regurgitation of food in the 1st 24 hours.
- Choking, cyanosis & cough

3. O/E:

- **General** → dehydration, pneumonia.
- **Local:**
 - a. Distention of the abdomen with respiration (*If fistula of lower pouch*).
 - b. Scaphoid abdomen (*If blind lower end*)
- May be presented with associated anomalies (**VACTERL syndrome**)
 - Vertebral → Spina bifida.
 - Anal → Imperforate anus
 - Cardiac → Fallot tetralogy, VSD, ASD.
 - Tracheal → Tracheo-esophageal Fistula
 - Esophageal → Atresia
 - Renal → Polycystic kidney
 - Limb → Polydactyly

- ▶ **Def.:** failure of recanalization
- ▶ **Types:** the commonest type is Blind upper pouch & fistula of lower pouch (87%)
- ▶ **C/P:** pouring of saliva (pathognomonic), regurgitation, VACTERL syndrome, comp.
- ▶ **Invest.:** catheter test, Gastrografin (the best)
- ▶ **TTT:** a. pre-operative
b. operative: with fistula, without fistula

DD

- **Posterior choanal atresia** → Arrest at 2 cm from nostril.

Causes of Death

- Pneumonia (always occurs).
- Associated anomalies (VACTERL syndrome).

Investigations

A. For Diagnosis

1) Instrumental:

- **Catheter test:** (*Routine check of newborn*)
→ It will be arrested usually **10 cm** from the nostril.

2) Radiological:

- **Plain X-Ray:**
 1. ↑ fundic air bubble → fistula of lower pouch.
 2. Absent fundic air bubble → blind lower pouch.
 3. Chest: pulmonary complication.
- **Gastrografen (best):**
→ Shows fistula or atresia of upper pouch.



Gastrografen showing arrest of dye + dye seen in the lung

- ▶ Lipidol → causes lipoid pneumonitis.
- ▶ Barium → destructive to bronchial mucosa & may cause tracheal obstruction.

- **Fiberoptic pediatric esophagoscopy.**
- **Antenatal diagnosis by U/S:** → Dilated upper pouch

3) Laboratory:

- **CBC, ABG** → to assess general condition of the patient.

B. For Associated Anomalies

e.g. Invertogram for imperforate anus, echo.

Treatment

Preoperative

- 1- NPO (Nothing per os).
- 2- Suction of saliva.
- 3- IV fluids.
- 4- Care of respiration.
- 5- Prone position (↓ aspiration) "any new born diagnosed as esophageal atresia should be kept in prone position".
- 6- Avoid hypothermia.

- ▶ It is an emergency which should be managed on the first day of life.
- ▶ Urgent surgery in pediatric center after preoperative care (before breast feeding starts)

Operative

A- Atresia with fistula :

- Rt. thoracotomy at the level of 5th Rt. rib (extrapleural approach).
- Excision of the fistula with end to end **anastomosis**.

B- Atresia without fistula:

- Usually there is a long gap which makes middle anastomosis difficult. Gastrotomy is done at birth.
- Further surgery to restore esophageal continuity is done 6 months later

+ Treatment of associated congenital anomalies

Diaphragmatic Hernia

Congenital Diaphragmatic Hernia

Definition

It is a herniation of some of abdominal contents through a congenital defect in the diaphragm.

Incidence

1: 4000

Embryology

1. Fusion of septum transversum and the pleuroperitoneal folds occurs bilaterally during the 8th week of intrauterine life (right side before left side).
2. Intestine returns to abdomen for rotation and fixation at 10th week.

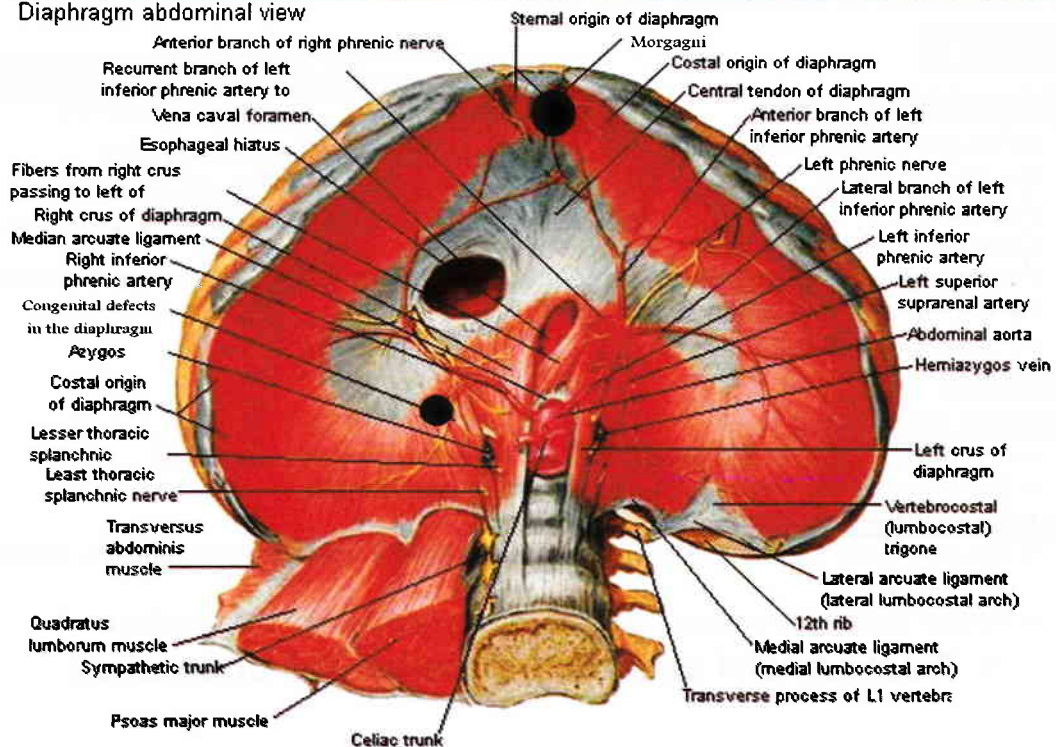
Types

➤ Main types of CDH:

- 1- Hernia through foramen of Bochdalek (posterior hernia) (**3P**): 90% of cases.
Left:right = 5:1
- 2- Hernia through foramen of Morgagni (anterior hernia): through opening of internal mammary artery.
- 3- Eventration of diaphragm (not a true hernia) due to weakness of part of diaphragm.
- 4- Hiatus hernia

- ▶ Bochdalek is the commonest diaphragmatic hernia in infants.
- ▶ Sliding H.H is the commonest diaphragmatic hernia in adults.

Diaphragm abdominal view



Presentation

Antenatal Diagnosis:

- Maternal polyhydramnios.
- Antenatal U/S is diagnostic

► Any newborn with cyanosis in absence of heart disease and a scaphoid abdomen is considered congenital diaphragmatic hernia until proved otherwise

At birth:

Symptoms

- Baby presents soon after birth with cyanosis & dyspnea.

Signs

- Scaphoid abdomen.
- Dull note over the chest.
- Diminished air entry or its absence on the ipsilateral side of the chest.
- Presence of intestinal sounds on the ipsilateral side of the chest.
- The heart sounds are better heard on Rt. Side in case of the common left sided hernia.

DD

Of neonatal respiratory distress

- Hyaline membrane disease.
- Esophageal atresia with aspiration.

Complications

- Ipsilateral pulmonary hypoplasia → pulmonary hypertension → leads to Rt. To Lt shunt.
- Contralateral compression of lung.
- Strangulation of contents.

Investigations

1. Radiological:

Plain X-Ray:

► It will demonstrate:

- Presence of gas shadow of the stomach or bowel in the chest cavity.
- Shift of trachea and the mediastinum to the contralateral side.
- Apparent dextrocardia.

Gastrografin meal:

- Presence of stomach and intestinal loops within chest cavity.

Antenatal diagnosis by U/S and Echo:

- May be used prenatal, it will show bowel in chest cavity.

Echo for associated cardiac anomalies and degree of portal hypertension.

2. Laboratory:

Blood gases:

- Hypoxia, hypercapnea, acidosis



Portions of intestine and spleen in the left chest with the heart displaced into the right chest



Treatment

Surgical treatment is the rule.

Preoperative Preparation (in incubator)

1. Nasogastric tube (NGT) to decompress the stomach.
2. Mechanical ventilation.
3. Inhaled NO to decrease portal hypertension.

► Never to use mask oxygenation.

Operation

- Through abdominal approach.
- The herniated contents are reduced & the defect is closed directly or a mesh is used if the defect is too big.

Postoperative

Continue ventilatory support

► **Recent trends:**

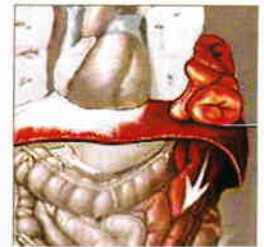
- Intrauterine surgical correction.
- Nitric oxide may be used to induce pulmonary VD.

(A)



(B)

Intestine is pulled back down to abdomen



(D)



(C)



Reduction of the hernia with repair of the diaphragm

Reflux Esophagitis (GERD)

Definition

- It is the reflux of acid juice (gastric contents) into the lower part of the esophagus.

- **Etiology:** Sliding H.H. (most important)
- **C/P:** heart burn, regurgitation, dysphagia, water brush, comp.
- **Comp.:** stricture, ulceration, Barrett's esoph.
- **Invest.:** 24 h. PH monitoring, manometry
- **TTT:** conservative (main line), surgical ttt

Normal anti-reflux measures

- **Angle of Hiss** (acute) → valvular effect between esophagus & stomach
- **Intra-abdominal esophagus** (lower 5 cm) → closed by intra-abdominal pressure
- **Phrenoesophageal ligament** → keeps intra-abdominal part in place
- **Rosette shaped** mucosa of lower end of the esophagus (cardia).
- **Pinchcock effect on esophagus** → by contraction of right crus of diaphragm
- **Continuous release of Acetyl-choline** from lower end of esophagus (only relaxes on swallowing) → lower esophageal sphincter is physiological sphincter.
- **Esophageal acid clearance depends on:**
 - Volume of refluxed material (**GE reflux is physiological; it only becomes pathological when it is excessive, resulting in symptoms or complication**)
 - Salivary secretions: rich in alkali which neutralizes the acid.
 - Gravity.
 - Esophageal motility.

Causes

1. **Sliding hiatus hernia** (most important).
2. **Obesity** → ↑ intra-abdominal pressure.
3. **Smoking & alcohol** → pylorospasm & hyperacidity.
4. **In infant** due to ↓ gastrin → ↓ tone of cardia (no fear of gastritis due to ↓ acidity).
5. **Scleroderma**.
6. 2nd due to pyloric stenosis or pylorospasm.

Pathology

“Belsky endoscopic grading of reflux:”

1. Grade I hyperemic mucosa.
2. Grade II intermittent superficial ulcers.
3. Grade III extensive ulceration.
4. Grade IV stricture or Barrett's esophagus.

➤ **Factors promoting reflux and damage:**

A- **Dysfunction of cardio-esophageal junction:**

1. Primary weakness of the lower esophageal sphincter.
2. Short length of the intraabdominal esophagus.
3. Abnormal high number of transient lower sphincter relaxations.

B- **Gastric distention:**

- Impaired gastric emptying whether organic (gastric outlet obstruction) or functional (due to food which slows gastric emptying like fat) may contribute to reflux.

Clinical Picture

Symptoms

Obese female > 40 years old with Heart burn related to fatty meal and lying flat

1. **Heart burn:**
 - Retrosternal burning pain simulating angina pain.
 - Increase by heavy meals (fatty food) and lying flat.
 - Decrease by standing upright.
2. **Regurgitation:** especially on lying down, relieved by sitting.
3. **Dysphagia** due to esophageal spasm at first, but later due to stricture formation.
4. **Water brush:** regurgitation of acid on lying flat or bending forward.
 - it is excess salivary secretion to neutralize acid regurgitation on lying or bending forward which presented by sudden filling the mouth with excess saliva
5. **Symptoms of complications (late):**
 - a. Aspiration pneumonia.
 - b. Bleeding from ulcer → anemia.
 - c. Barrett's esophagus (columnar epithelium lining the esophagus) → precancerous.
 - d. Esophageal stricture (Schatzki's ring).
 - e. Hoarseness of voice and loss of dental enamel.

► Fat dyspepsia is more common in GERD than Gall stone disease.

Signs

General → Bad nutritional state.
→ Chest infection, anemia.

Complications

1. Stricture and shortening “Schatzki's ring”.
2. Deep ulcerations → anemia.
3. Columnar metaplasia “Barrett's esophagus”. (↑ risk of adenocarcinoma 25 times)

► **Factors associated with development of complications of GERD:**

1. Presence of defective LES.
2. Presence of HH.
3. Inadequate esophageal clearance.
4. Presence of alkaline component in reflux material.

Investigations

A-For diagnosis:

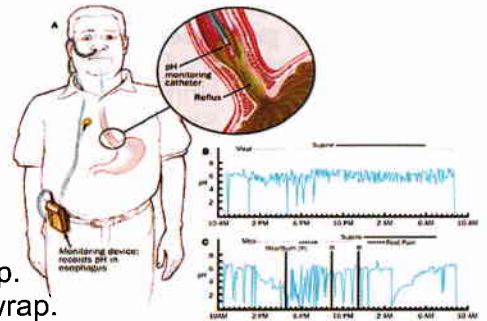
1. Instrumental:

➤ 24 hour ambulatory pH monitoring:

- pH < 4 in distal 5 cm of esophagus above the high pressure zone = clear evidence.
- Most diagnostic findings: pH < 4 for more than 30 min in 24 hr.

➤ Esophageal manometry:

- It assesses the LOS pressure and peristalsis.
- It helps to choose the proper surgical procedure, how?
 - Weak peristalsis → partial wrap.
 - Good peristalsis → complete wrap.
- It will reveal loss of high pressure zone of the lower end of esophagus in case of sliding hiatus hernia.



➤ Radioisotope Scintigraphy.

➤ Upper GI endoscopy (biopsy + cytology in doubtful cases → Belsy's grading) for columnar metaplasia "Barret's esophagus"

- Cardia opens on inspiration (normally closed).
- Reflux will be revealed.
- Complications (esophagitis, ulceration or stricture).

2. Radiological:

➤ Barium swallow & meal in 20° (Trendelenburg position):

- Reflux of barium from stomach to esophagus.
- In case of sliding hiatus hernia: → part of stomach in the chest (gases in chest).

B-For complications:

- CBC → microcytic hypochromic anemia due to chronic blood loss.

Treatment

I. Conservative (Main line):

➤ Life style modification:

- Reduction of weight.
- Small frequent meals.
- Avoid tea, coffee, spirits & smoking.
- Avoid lying flat especially after meals.
- Sleep with extra pillows.
- Avoid constricting clothes as corset.

- (1) PPIs are the most effective single drug for esophagitis.
- (2) Prokinetics are as effective as H₂ blockers.

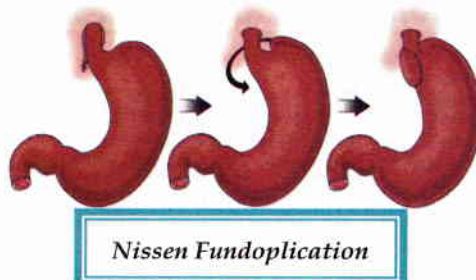
- **Drug Therapy: (Prokinetic drugs)**
 - **Drugs to decrease gastric acidity:**
 - **Antacids:** for mild cases.
 - **H₂ blockers:** ↓ gastric acid production; Ranitidine (Zantac™) 300 mg at bed time.
 - **PPI:** Omeprazole, Lansoprazole (*reverse Barret's esophagus*)
 - **Drugs regulating motility:**
 - ↑ Lower esophageal sphincter tone.
 - Accelerate gastric emptying.
 - **e.g.** Metoclopramide, Cisapride (*not used, arrhythmogenic*).

II. Surgical:

Approximately 10-15% of patients with GERD will be referred for consideration to have antireflux surgery.

- **Indications:**
 1. Failure of conservative treatment after 6 months.
 2. Presence of complications:
 - Hemorrhage or chronic anemia.
 - Esophageal stricture with failure of endoscopic dilation.
 - Respiratory complications.
 3. Presence of other indications for laparotomy e.g. Saint's triad.
 4. Non compliance of the patient.

- **Procedure:**
 - Nissen fundoplication includes a complete wrap of the fundus of the stomach around the lower end of the esophagus (360°) to create a high pressure zone.
 - This operation can be done by open or laparoscopic surgery.



Acquired Diaphragmatic Hernia

HIATAL HERNIA

- **Type 1:** Sliding HH is the most common diaphragmatic hernia in adult and always presented as GERD. Its main line of treatment is conservative.
- **Type 2:** Rolling hernia is the most dangerous type of hernia. Surgical treatment is the rule.
- **Type 3:** mixed hiatal hernia.

Sliding Hiatus Hernia

Definition

- Herniation of the cardia and adjoining part of the stomach through the esophageal hiatus in the diaphragm into posterior mediastinum.

Causes

- Increased intra-abdominal pressure as in pregnancy, obesity or wearing of tight corsets.
- Decreased elasticity of the right crus of the diaphragm as in obesity or old age.

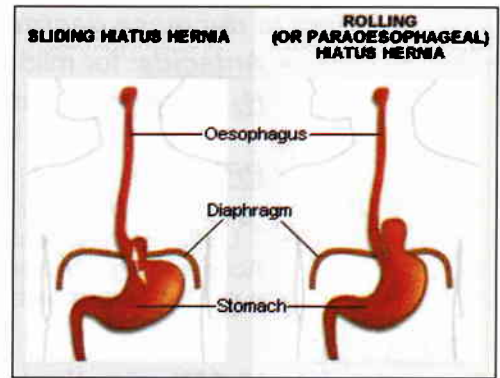
- ▶ Commonest of all internal hernias.
- ▶ **Def.:** herniation of the cardia through the diaphragm → loss of the high pressure zone
- ▶ **C/P:** (obese ♀ > 40 years) asymptomatic, GERD, comp., saint's triad
- ▶ **Invest.:** Barium meal in the Trendelenburg's position
- ▶ **TTT:** only if presented by GERD

Incidence

- Commonest of all internal hernias.

Pathology

- The gastroesophageal junction is displaced into the chest with stretch of phrenoesophageal ligament → loss of the high pressure zone → reflux of acid → esophagitis and ulceration → fibrosis → more pulling into the chest
- Herniation is associated with a small empty peritoneal sac on the left side of the stomach.
- Prolonged esophagitis leads to replacement of esophageal mucosa by columnar cells (Barret's esophagus). It develops in 10% of cases of GERD.
- It is a precursor for ulcers, dysplasia, cancer in situ and adenocarcinoma.



Clinical Picture

Symptoms

Obese female > 40 years old

1. Usually asymptomatic.
2. If the episodes of reflux → increase the patient will have the clinical picture of GERD
3. Symptoms of complications:
 - As in GERD
4. May be part of Saint's Triad:
 - Hiatus Hernia (HH)
 - Diverticular disease of the colon (DD)
 - Chronic calculous cholecystitis (CCC)

Signs

General: - Bad nutritional state. - Chest infection.

Investigations

1. Barium meal in the Trendelenburg's position:

- The hernia appears as a small epiphrenic bulge.
- Widening of the esophageal hiatus.
- Usually there is reflux of barium from the stomach to the esophagus.
- If there is esophagitis, there will be irregularity of the esophageal lumen with granular mucosal pattern.

2. Perform the investigations of GERD: (see before).

Treatment

- ➔ It should be stressed that sliding hiatus hernia per se does not need treatment.
- ➔ Only if the symptoms of GERD are severe, then surgery will be needed.
- ➔ The principles of surgery are:
 - Reduction of the hernia.
 - Maintain a segment of the esophagus intra-abdominally.
 - Performance of antireflux mechanism (Nissen's fundoplication).
 - Repair of the right crus of the diaphragm.

Paraesophagea(Rolling) Hernia

Pathology

- 1- It is a true ☒ hernia in which the greater curvature of the stomach herniates into the posterior mediastinum within a preformed peritoneal sac.
- 2- Normal relationship of cardiesophageal junction to diaphragm is undisturbed ☒ → no reflux.

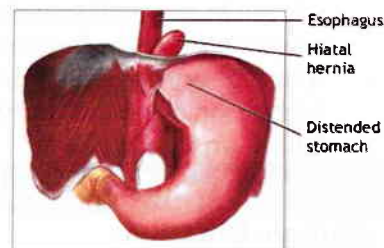
Clinical Picture

Small Asymptomatic.

Large

➤ Compresses:

- Esophagus → intermittent dysphagia
- Heart → postprandial chest pain and palpitation.
- Phrenic nerve → bouts of hiccough.



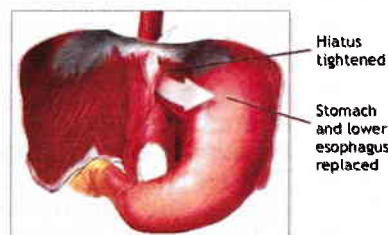
Complications

Life threatening

1. **Rupture** → mediastinitis.
2. **Strangulation** → gangrene

Investigations

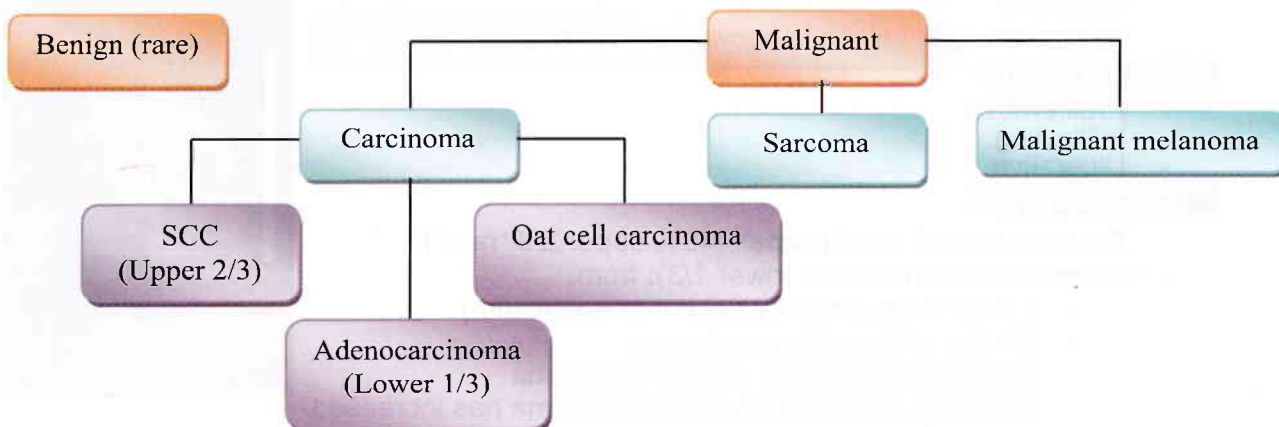
- 1- **Plain X-Ray:** Show gastric gas shadow in chest
- 2- **Barium meal:**
 - In Trendelenburg position → herniation of the stomach into chest, esophagogastric junction in its place



Treatment

- Stomach is retracted downwards; hernial sac is excised with repair of hernial defect in the diaphragm.

Tumors of the Esophagus



Esophageal Carcinoma

Incidence

- **4% of GIT tumors.**
- **Age** → 45-60 years old.
- **Sex** → Male (5% of all carcinomas) except in cervical esophagus may be more common in females.
- It has geographical distribution showing higher incidence in some countries as China and Japan.

- ▶ **PDF:** Chronic irritation, benign tumors
- ▶ **C/P:** Old male with rapidly progressive dysphagia to solid > fluids
- ▶ **Comp.:** direct spread + pulmonary comp.
- ▶ **Invest.:** **a. diag.:** -Esophagoscopy + biopsy
-Barium swallow: Rat tail appearance
- b. staging** **c. preop. preparation**
- ▶ **TTT:** **a.** mostly inoperable (palliative TTT)
b. If operable surgical excision

Predisposing Factors

⇒ Chronic Irritation:

- Spicy food, smoking & spirits.
- Food contamination by fungi
- Niroamine used in food preservation.
- **Barrett's esophagus** which is lined by columnar epithelium in reflux → adenocarcinoma.
- Achalasia of esophagus.
- **Tylosis type A** (A.D, characterized by hyperkeratosis of palm and sole, 100% esophagus carcinoma at age of 40 years).
- ↓ β carotene, selenium, vitamin E in diet.
- **Plummer-Vinson syndrome.**
- Postcorrosive.
- Scleroderma.

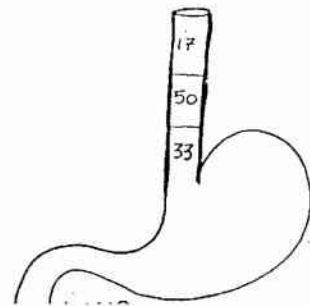
⇒ Benign tumors: Papilloma or adenoma.

Pathology

Site

- Upper 1/3: 20%
- Middle 1/3: 30%
- Lower 1/3: 50%

▶ Nowadays lower 1/3 is the most common

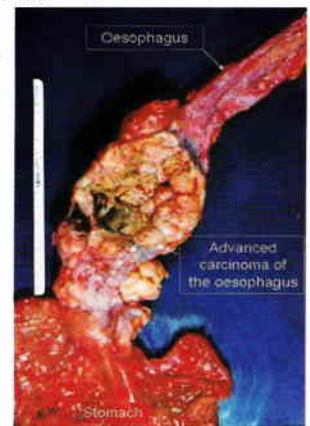


Macroscopic

- Proliferative.
- Infiltrating.
- Ulcerative.

Microscopic

- **Squamous cell carcinoma** (40% upper 2/3 "rare").
- **Adenocarcinoma** (60% lower 1/3): from;
 - Lower 3 cm (lined by columnar epithelium)
 - On top of Barrett's esophagus.
 - Upward spread from gastric carcinoma
 - Recently incidence of adenocarcinoma has increased.
- **Anaplastic:** the cells show malignant criteria (poorest prognosis).



Spread

Direct spread (Most DANGEROUS)

1. Spreads transversely then longitudinally (extensive submucosal lymphatic spread, proximal line of resection should be 10 cm proximal to the upper limit of the tumor).

2. Neighboring structures:

- Cervical esophagus:
 - Trachea → fistula.
 - Recurrent laryngeal nerve → vocal cord paralysis.
 - Thyroid.
- Thoracic esophagus:
 - Trachea → fistula.
 - Aorta → fatal hematemesis.
 - Left recurrent laryngeal nerve.
 - Lung.
 - Thoracic vertebrae.
- Abdominal esophagus:
 - Liver.
 - Stomach.
 - Diaphragm.

Lymphatic Spread (early)

- Cervical esophagus → deep cervical LNs.
- Thoracic esophagus → posterior mediastinal, tracheal & tracheobronchial LNs
- Abdominal esophagus → left gastric & celiac LNs..

Blood Spread

- Rare & late.
- Occurs mainly to liver & lungs.

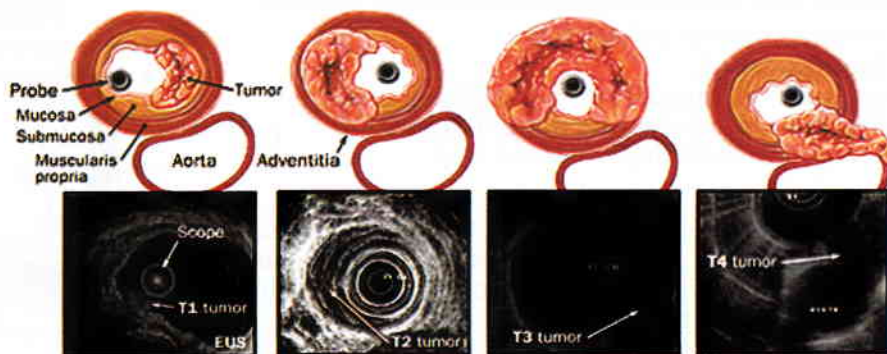
Transcoelomic

- Only in abdominal esophagus:
 - ⇒ When the tumor reaches the serosa → malignant cells spread toward the peritoneal cavity → peritoneal nodules + malignant ascites then to pelvis → **Krukenburg tumor** + nodules in Douglas pouch (Plummer shelf).

Staging

TNM Classification

T = Primary tumor	N = Nodes	M = Metastasis
T_{is} = carcinoma in situ T₁ = tumor is confined to esophageal mucosa T₂ = tumor reaches esophageal muscosa T₃ = any size with extraesophageal spread T₄ = invades adjacent structures	N₀ = No LN enlargement N₁ = Mobile unilateral L.N affection N₂ = Mobile bilateral L.N affection N₃ = Fixed LNs	M₀ = no evidence of distant metastasis M₁ = distant metastasis



T staging of cancer esophagus with corresponding endoscopic U/S images

Clinical Picture

Symptoms

Old male with rapidly progressive dysphagia to solid > fluids

- ▶ **Cancer esophagus has poor prognosis and poor survival rate due to late occurrence of symptoms and late discovery after spread.**

- **Male > 50 years old .**
- **Dysphagia: (indicates advanced stage of the disease)**
 - Continuous rapidly progressive dysphagia to *solids* > *fluids*[□], but later the patient cannot swallow his own saliva leading to continuous dripping of saliva.
 - Usually, at first presentation about $\frac{2}{3}$ of circumference of esophagus is already involved.
 - Swallowing may become easier due to sloughing of a portion of the tumor (may occur 2-3 times).
- **Regurgitation** (blood stained) leads to pulmonary symptoms.
- **Excessive salivation.**
- **Appetite is finally lost.**
- **Hematemesis is not common.**
- **Symptoms due to infiltration of adjacent structures e.g. change of voice due to infiltration of RLN.**

Signs

- **General:**
 - Cachexia, dehydration & chest infection.
- **Local:**
 - Neck: for presence of lymph nodes or mass.
 - Abdomen: for palpable hard nodular liver & ascites.

Complications

- 1- **Mediastinitis:** due to esophageal perforation.
- 2- **Fatal hematemesis:** due to aortic invasion.
- 3- **Paralysis** of diaphragm and vocal cords.
- 4- **Pulmonary complications:** (pneumonia, lung abscess etc.) (cause of death)

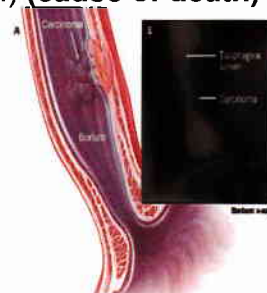
Investigations

1. For diagnosis:

- Esophagoscope “**early endoscopy is the key for good result**” + biopsy + cytology
- Barium swallow:
 - **Rat tail appearance**[□]: Narrow lumen at site of lesion with mild proximal dilatation due to short duration (unlike achalasia)
 - **Shouldering**[□].
 - **Irregular filling defect** in cauliflower mass.
 - Peristaltic wave may be absent above the lesion due to infiltration of the wall .

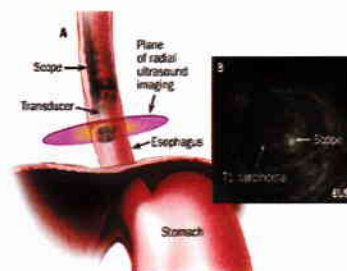
2. For staging:

- Endoluminal sonar: show extent of tumor (the most important for local staging and assessing operability)
- Chest X-Ray: elevated cupola of diaphragm due to affection of phrenic nerve, pleural effusion.
- U/S → liver metastasis.
- CT scan.
- Bone Scan.



A. Esophageal carcinoma;

B. corresponding barium swallow x-ray.



A. Radial scanning for esophageal carcinoma;

B. corresponding endoscopic ultrasonography

- Bronchoscope → invasion of the trachea (bronchoscope is done before barium swallow because if there is fistula → barium will pass into trachea).
- Indirect Laryngoscope → invasion of recurrent laryngeal nerve.

3. For preoperative preparations:

- CBC: anemia & leucocytosis in chest infection.
- LFTs: for metastasis. - KFTs.
- Serum electrolytes & serum protein

Treatment

Most cases are inoperable at time of presentation.

1. Inoperable Tumors (60% of the patients)

⇒ **Signs of Inoperability:-**

➤ **Clinical:**

1. Distant secondaries.
2. Enlarged LNs.
3. Voice change → recurrent laryngeal nerve infiltration.

➤ **Radiological:**

1. Enlarged mediastinal LN s
2. Diaphragmatic paralysis.
3. Vertebral erosion.

➤ **Bronchoscopic:** Tracheal invasion.

➤ **Exploratory:**

1. Fixed tumor.
2. Aortic invasion.
3. Secondaries in liver.

⇒ **Treatment:**

➤ Bypass:

- Gastric bypass with a cervical esophagogastrostomy.
- Colon bypass.
- Major operation with high mortality for a patient with short life span

- **Radiotherapy:** *(suitable for carcinoma in upper esophagus)*

- The dose of X-Ray should be between 4000 and 4500 rads / 4 wks
- Patients treated with high doses complicated with; pulmonary fibrosis, esophageal bleeding or esophageal perforation.
- Suitable for upper esophagus

➤ **Intubation:**

- The idea is to insert a rigid tube through the stenosed segment to keep a patent lumen.
- The tube is inserted by :
 - o Gastrostomy (e.g. Celestin tube)
 - o Esophagoscopy (e.g. Souttar tube).

➤ Laser photocoagulation (YAG):

- Dysphagia can be relieved by endoscopic laser therapy.
- A core of tumor is vaporized, opening the lumen without perforating the esophagus.
- Treatment needs to be repeated every 6-8 weeks.

➤ **Gastrotomy:**

- Obsolete nowadays as the patient remains unable to swallow his saliva
→ aspiration pneumonia

➤ **Chemotherapy:** 5FU.

2. Operable tumors

⇒ Indications:

- Good general condition
- No advanced local or distant spread.

⇒ Preoperative Preparation:

- Nutritional Status: these patients often need hyperalimentation.
- Respiratory System: Respiratory exercises, treatment of chest infection.
- Hematological: Correction of Hb, Albumin
- ✓ Idea of the operation: To resect the lesion with adequate safety margin in either side (10 cm) & then restore the continuity of the GIT

⇒ Surgery:

1) Tumors below the carina (tracheal bifurcation):

Ivor Lewis operation (2 phases) for middle 1/3 tumors;

- 1st phase: Laparotomy & mobilization of stomach.
- 2nd phase: Rt thoracotomy through the 5th intercostal space, resection of the tumor, LNs and 10cm of the esophagus above the tumor & GE anastomosis.

2) Tumors above the carina:

Mc Keown operation (3 phases)

- 1st phase: Laparotomy & mobilization of stomach.
- 2nd phase: Rt thoracotomy through the 5th intercostal space and oesophageal mobilization.
- 3rd phase: Neck incision, the esophagus & stomach are delivered to the neck where resection is done and anastomosis of the stomach & cervical esophagus is carried out.

3) Tumors below the diaphragm (1 phase) for lower 1/3 tumours

- Lt thoracoabdominal incision, the stomach & lower esophagus are removed with Roux-en-Y esophagojejunostomy.

4) Nowadays many surgeon prefer to do:

Transhiatal total esophagectomy

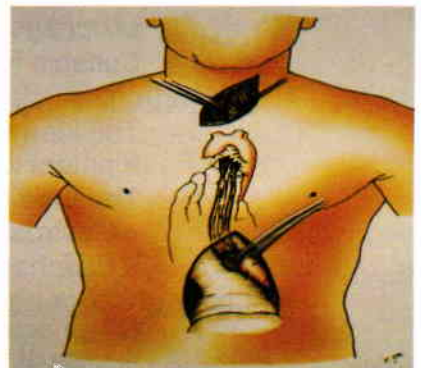
- Thoracotomy is avoided by mobilizing the esophagus from the abdomen via the diaphragmatic hiatus and via the neck incision.

➤ Advantages:

- No need for thoracotomy.
- Anastomosis in neck (if leakage occurs, it is not dangerous).
- Guarantee of an adequate safety margin.
- Stomach can be mobilized easily.

Endoscopic removal

- Through laparoscopy & thorascopy



⇒ Esophageal Replacement surgery

1) **Stomach pull up is superior to colon bypass** due to:

- ❖ **More** anatomical & physiological.
- ❖ **Good** blood supply.
- ❖ **Single** anastomosis (colon 3 anastomosis).

2) **Colon replacement:** a segment of Rt or Lt colon is mobilized with preservation of the feeding artery & vein.

The colon is anastomosed below to the stomach and above to cervical esophagus

3) **Pectoralis major myocutaneous flap:** Can be used to replace a localized segment of the cervical esophagus

4) **Free jejunal replacement with microvascular anastomosis**

- *Recently surgeons prefer transhiatal esophagectomy to avoid opening the chest*
- *Transhiatal esophagectomy can replace McKeown's to avoid thoracotomy.*
- *Ivor Lewis is not highly popular as leakage of chest anastomosis is fatal.*

➤ Postoesophagectomy complication:

Reflux (Can be avoided by subtotal esophagectomy & gastric transposition high in the chest)

➤ Neoadjuvant treatment (Chemoradiotherapy.)

Can be effective with surgery especially with squamous cell carcinoma

Bailey & love's 25th edition chapter 59 P. 1034, 1035

- ▶ **Ba swallow** → esophagus.
- ▶ **Ba meal** → esophagus, stomach, 1st part of duodenum.
- ▶ **Ba enema** → rectum, large intestine.
- ▶ **Ba Meal follow through** → esophagus → anus.

Barrett's esophagus

- 10% of cases of GERD.
- Metaplasia of the lower end of the esophageal mucosa into columnar type.
- A precursor for ulcers, dysplasia, cancer in situ and adenocarcinoma.
- Regular endoscopic monitoring with multiple biopsies is essential.
- Endoscopic mucosa resection (EMR) using photodynamic therapy or argon beam coagulation may be used before progression to cancer.
- The best line of TTT of it is large dose of PPIs

Postcricoid carcinoma

Definition

- Carcinoma of hypopharynx at the level of cricoid cartilage.

Etiology

- It may occur on top of **Plummer-Vinson** syndrome.

Pathology

Site

- Pharyngeal mucosa

Macroscopic

- Usually fungating mass.

Microscopic

- Squamous cell carcinoma.

Spread

Direct

- **Forward** → larynx.
- **Lateral** → pyriform fossa.
- **Downward** → esophagus.

Blood

- Mainly to the lung.

Lymphatic

- To deep cervical LNs.

- ▶ **Etiology:** Plummer-Vinson syndrome
- ▶ **C/P:** throat pain, late dysphagia, Moore's sign
- ▶ **Comp.:** local invasion, obst., metastasis
- ▶ **Invest.:** endoscopy + biopsy
- ▶ **TTT:** operable, inoperable

Clinical Picture

Symptoms

- Pain in the throat referred to the ear through **Arnold's nerve** [☐].
- Dysphagia (**late**) [☐].

Signs

- **Inspection:** bulge of thyroid cartilage and trachea
- **Palpation:** loss of laryngeal click (**Moure's signs**) [☐]; which is felt when larynx is moved from side to side over the vertebral column

Complications

1. Obstruction:
 - Esophageal → dysphagia.
 - Laryngeal → stridor and dyspnea.
2. Hoarseness of voice.
3. Ulceration, hemorrhage & infection.
4. Metastasis.

Investigations

1. For diagnosis:

- **Pharyngoscopy and laryngoscopy:**
 - Visualize the tumor
 - Can take biopsy from the tumor.
- **Plain X-Ray:**
 - Widening of space between pharynx and larynx.
- **Barium swallow:**
 - Filling defect in pharynx

2. For staging:

- C.T, CXR, Bone scan,.....

3. For Preoperative preparation:

- CBC, KFTs, LFTs

Treatment

Operable

- Total laryngopharyngectomy + stomach pull up with block dissection of LNs + permanent tracheostomy and esophageal replacement.

Inoperable

- Radiotherapy.

Pharyngeal Pouch, Zenker's Diverticulum, Pharyngoesophageal Diverticulum

Definition

- Herniation of pharyngeal mucosa through a weak area in the posterior pharyngeal wall (**Killian's dehiscence**)[☐]; between upper oblique fibers (thyropharyngeus ms.) and lower transverse fibers (**cricopharyngeus ms.**) of inferior constrictor muscle of the pharynx.
- It is acquired, pulsion diverticulum.

- **Etiology:** spasm of cricopharyngeus ms
- **C/P:** dysphagia, n.swelling, regurgitation
- **Comp.:** pulmonary, perforation, pouch inflammation, pouch malignancy
- **Invest.:** Barium swallow
- **TTT:** acc. to size (large → diverticul-ectomy + cricopharyngeal myotomy)

Pathology

- Spasm of cricopharyngeal ms. → increase pressure in the pharynx → herniation of mucous membrane posteriorly (**pulsion diverticula**) till limited by spine → then laterally (usually) **to the Lt. side**[☐] due to inclination of carotid vessels → enlarges taking vertical line → displacing esophagus → so food enters the pouch more readily than esophagus and risk of perforation by endoscope.

Clinical Picture

► **Old male with dysphagia and swelling in the neck, when he compresses this swelling regurgitation of undigested food occurs**

- Old male, with progressive dysphagia.
- Regurgitation of non digested food (non acidic) especially lying on one side.

O/E: (swelling)

- On the Lt. posterior triangle.
- ↑ with eating.
- Soft & compressible.
- Dull or resonant on percussion.
- Gurgling sound can be detected if patient swallows large amounts of air.

Complications

- Pulmonary complication → inhalation → pneumonia and lung abscess.
- Perforation with endoscope.
- Diverticulitis.
- Carcinoma (0.3%)**[☐]

Investigations

- Ba swallow (**safest**):
 - Early there is a bulge in the posterior wall of the pharynx then flask shaped pouch
- Endoscope (**may lead to perforation**)[☐].

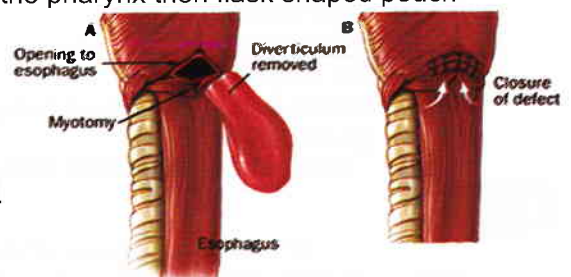
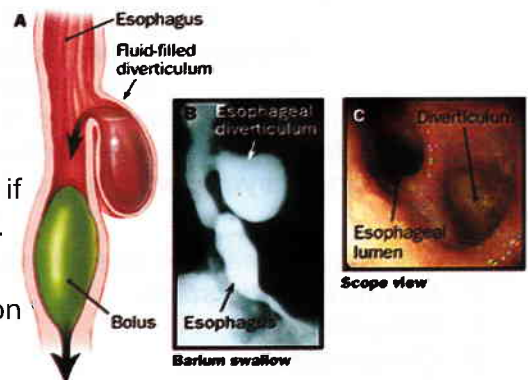
Treatment

According to Size

- Small → repeated dilatation.
- Large → diverticul-ectomy, **cricopharyngeal myotomy** to avoid recurrence.

Old, Poor Risk Patient

- Remove wall between diverticulum and esophagus by endoscopy, laser photocoagulation.



Achalasia of the Esophagus (Cardiospasm)

Definition

- Functional disorder of esophagus characterized by **failure of relaxation** of the cardia during swallowing & **absent primary peristalsis** above it associated with dilatation and hypertrophy of the circular muscle layer.

- **Etiology:** unknown (autoimmune ?)
- **C/P:** dysphagia (intermittent & towards fluids > solids), halitosis, bad chest
- **Invest.:** Ba swallow, manometry, comp.
- **TTT:** medical, forced dilatation, surgical (Modified Heller's operation)

Etiology

- Degeneration of **Auerbach's plexus**, vagal fibers or nucleus ambiguus.
- The inhibitory neurons are selectively affected.
- The exact cause is **unknown**.
- May be autoimmune

Clinical Picture

Type of patient

- More often in 2nd to 4th decades.
- Equal in both males & females.

Symptoms

- Dysphagia** → -Gradual onset, intermittent course & of long duration.
-Towards fluids > solids.

Postural Regurgitation → alkaline, foul smelling
(when patient lies down during night)

- Halitosis** due to stasis of food → putrefaction.
- Retrosternal pain** (esophagitis) (stasis → infection).
- Pulmonary symptoms:** as aspiration & wheezing.



Signs

General (present in late cases only)

- Bad nutritional state.**
- Dehydration.**
- Chest infection.**
- Toxic rheumatoid arthritis**

Complications

- Aspiration pneumonia.**
- Diverticulae** of esophageal wall.
- Malnutrition** → anemia & hypovitaminosis.
- Toxic arthropathy.**
- Malignant changes**☑:
 - 3% after 20 years.
 - Discovered late → bad prognosis, as the patient already has dysphagia due to achalasia.
 - Treatment of achalasia **will not ↓ incidence** but allows early discovery.

DD

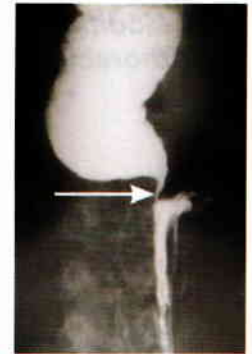
(From other causes of dysphagia mainly from carcinoma)

Old ♂, rapidly progressive dysphagia of short duration & more to solids.

Investigations

I. For diagnosis:

- **Esophageal manometry:**
 - Weak peristaltic wave
 - Failure of relaxation of LOS.
 - Pressure in high pressure zone is > 25 mmHg (N=8-25 mmHg).
- **Barium swallow) main diagnostic tool:**
 - Early → some barium remains held up (delayed evacuation).
 - Esophagus appears dilated and tortuous → **sigmoid esophagus**.
 - Lower end is pointed (narrow) → **[Parrot (Hen's)- beak appearance]**
 - Absence of air in the fundus of stomach



In barium meal if the esophagus is interrupted above the diaphragm it is mostly carcinoma of lower end esophagus but if the cardia below diaphragm is narrowed it is mostly achalasia.

- **Plain X-Ray chest:**
 - **Absence of gastric air bubble.**
 - Widening of mediastinum.
 - Pneumonia.
 - Lung abscess.
- **Esophagoscopy:** main aim is to exclude carcinoma: **(Biopsy + cytology in doubtful cases)**
 - Esophagus is wide, filled with dirty water.
 - Signs of esophagitis (red esophagus).
 - Narrow eccentric cardiac orifice (golf ball appearance) → esophagoscope can pass, but in carcinoma it is not wide enough for visualization.

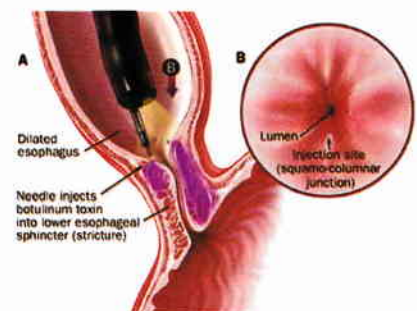
2. For complications:

- **CBC:** anemia, hemoconcentration, leucocytosis.

Treatment

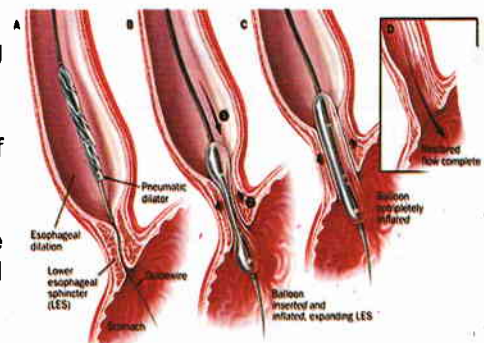
Medical Treatment

- **Isosorbide binitrate (nitrates)** or **CCBs** (usually ineffective).
- Endoscope injection of **Botulinum toxin:** tried to relax LOS (previously tried in all spastic GIT diseases).



Forcible Dilatation by

- a) **High pneumatic pressure balloon:** using Regiflex tube.
- b) **Plummer's hydrostatic balloon: (obsolete)**
 - Done without anesthesia to avoid rupture of the lower part of the esophagus.
 - Introduced through esophagoscope.
 - Inflate the balloon to produce rupture in the circular muscle fibers to relieve the distal esophageal obstruction.
 - Usually effective in most cases (80%)

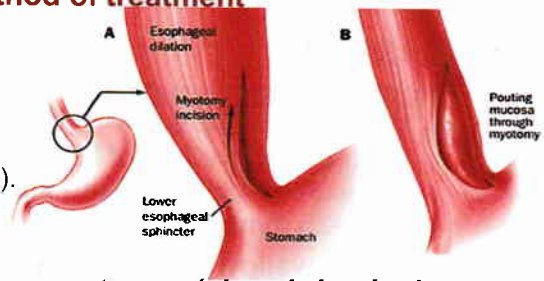


Surgery

Most reliable method of treatment

Indications

- Failure of all above measures.
- Advanced cases.
- Complicated patient.
- Uncooperative pt.
- Associated pathology (e.g. hiatus hernia).



Modified Heller's operation (Esophagomyotomy (via abdominal or thoracic approach)):

- **Myotomy is only esophageal:**
 - (Not extending to cardia) *depending on manometry.*
 - This is done to avoid reflux[□].
 - Expose the lower part of esophagus & cut muscle fibers completely until mucosa bulges through the incision.
 - Some authors advise antireflux mechanism

Diffuse Esophageal Spasm (DES) (Corkscrew Esophagus)

- ⇒ In this condition the normal peristaltic wave are replaced by simultaneous repetitive & occasionally prolonged increase in pressure in response to deglutition.
- ⇒ The main symptom is retrosternal pain rather than dysphagia.
- ⇒ **Treatment:** is by long myotomy from the aortic arch to the cardia.

Dysphagia

Definition

- Difficulty in swallowing (while odynophagia is painful dysphagia).

Causes

In the Mouth

- Stomatitis, glossitis, neoplasms, and ulcers of tongue and cheek.

In the pharynx

- Pharyngitis and tonsillitis.
- Retropharyngeal abscess.
- Plummer-Vinson syndrome.
- Pharyngeal diverticulum.
- Postcricoid carcinoma.

In the Esophagus

A. Mechanical causes:

⇒ Lumen:

- Foreign body.

⇒ Wall:

- Congenital stenosis.
- Traumatic as corrosive stricture.
- Inflammatory as reflux esophagitis.
- Neoplastic as carcinoma.

⇒ Compression from outside:

- Malignant thyroid.
- LNs.
- Thoracic aortic aneurysm.

B. Neuromuscular:

- Achalasia of the cardia.
- Neuritis of glossopharyngeal or vagus nerve.
- Myasthenia gravis, tetanus, rabies.
- Esophageal motility disorder e.g. corkscrew esophagus.
- Hysterical or bulbar palsy.

History

Personal History

⇒ Age:

- Child → F.B., postcorrosive.
- Young → achalasia.
- Old → carcinoma.

⇒ Sex:

- Male → carcinoma.
- Female → achalasia.

History of Present Illness

1. Dysphagia:

➤ Onset:

- Sudden → F.B., globus hystericus.
- Acute → inflammation.

➤ Course:

- Progressive → carcinoma.
- Intermittent → achalasia.
- Regressive → inflammation.
- Stationary → F.B., postcorrosive.

➤ Duration:

- Weeks to months → carcinoma.
- Years → achalasia.

➤ Type of food:

- More to fluids → achalasia (functional obstruction).
- More to solids → carcinoma (mechanical obstruction).

2. Regurgitation:

- Undigested food → pharyngeal pouch.
- Water brush → reflux.

3. Excessive salivation: in carcinoma (tongue, pharynx, esophagus).

4. Appetite: ↓ in malignancy.

5. Weight loss: in malignancy.

6. Retrosternal pain: → GERD.

7. Hematemesis: → GERD and malignancy.

Examination

A. General:

- State of nutrition, dehydration and anemia.

B. From above downwards:

- **Oral cavity:** inflammation, burns, tumor.
- **Neck:** LNs, laryngeal click.
- **Chest:** infection
- **Abdomen:** secondaries in liver.
- **Upper limb:** hypotension, tachycardia.
- **Lower limb:** edema (hypoalbuminemia).

Investigations

1. Laboratory:

- CBC → anemia (in malignancy and reflux).

2. Radiological:

1. Barium swallow:

- Rat tail appearance (cancer esophagus).
- Parrot beak appearance (achalasia).
- Hiatus hernia, pharyngeal pouch.

2. Chest X-Ray:

- Chest infection.
- Elevated copula of diaphragm in malignancy.

3. ECG → left atrial hypertrophy.

3. Instrumental:

1. Esophagoscopy + biopsy + cytology:

- Malignancy.
- Achalasia (dirty water, dilated esophagus, narrow eccentric cardiac orifice).
- Reflux (hyperemia, erosion, ulcer & stricture).

2. Bronchoscopy:

- Tracheal invasion.
- Vocal cord paralysis.

3. 24 hours ambulatory pH monitoring:

- Diagnosis of GERD.

4. Esophageal manometry:

- Esophageal motility disorders and hiatus hernia.

Treatment

(According to the cause) examples:

1. Achalasia:

- Medical: Ca channel blockers, isosorbide.
- Forcible dilatation.
- Modified Heller's operation.

2. Plummer-Vinson \$:

- Iron therapy.
- Dilatation through esophagoscope.

Traumatic Diseases of the Esophagus

I- Esophageal Perforation

Etiology

▪ Iatrogenic:

- Unskilled esophagoscopy, during removal of F.B. or stricture dilatation.
- The commonest site is its upper end.

▪ Accidental:

- FB, caustic ingestion and stab wound.

▪ Spontaneous:

- The condition is likely affect patients with head trauma and drunken
- in Both situations there is vomiting and uncoordinated esophageal motility
- The lower esophagus fails to relax in front of the ejected vomitus so the pressure markedly rises & the wall becomes stretched & perforates
- As a result the wall gives way either partially were the mucosa only is split producing severe bleeding (Mallory weiss syndrome) , or completely (**Boerhaave's \$**) through the whole wall thickness.
- The tear is usually situated in the lower posterior aspect

- ▶ **Etiology:** iatrogenic, accidental, spontaneous (Boerhaave's \$)
- ▶ **C/P:** Dysphagia, chest pain and Dyspnea
- ▶ **Invest.:** X-ray, gastrografen swallow
- ▶ **TTT:** acc. to site (cervical, thoracic)

Clinical Picture

- Severe dysphagia.
- Severe chest pain.
- Mediastinal emphysema appears as crepitus at the base of the neck, later subcutaneous emphysema over the chest wall & the neck.
- Dyspnea due to (pneumothorax and pneumomediastinum) & cyanosis.
- Fever and toxemia due to pleural empyema and mediastinitis.

Investigations

1- Plain X- Ray:

- Air in neck, pleura and mediastinum.
- Pleural effusion.

2- Esophagogram (Gastrografen Swallow):

- Using water soluble contrast medium
- Should be performed immediately in every patient suspected of having an esophageal perforation.
- The examination should be repeated using barium.

3- Thoracocentesis:

- Reveals cloudy or purulent fluid depending on how much time has passed.

Treatment

➔ Cervical perforation :

1. Nil by mouth
2. IV hyperalimentation
3. Drainage of the extravasated fluid
4. Intensive antibiotics
5. If the case is early , surgical closure of the perforation may be successful

➔ Thoracic perforation

1. If the diagnosis is early, suture of the perforation & chest drainage are often successful . A flap from the gastric wall may be utilized to close the perforation
2. If the diagnosis is delayed any attempt to close the perforation will fail. esophagectomy & a gastric pull up operation with chest drainage may save the patient .

II- Postcorrosive Esophageal Stricture

Etiology

- Ingestion of caustics accidentally or attempted suicide.

Pathology

- The extent of damage depends upon the concentration of chemical & the duration of tissue contact.
- Alkalies → immediate **liquefactive necrosis** of all esophageal layers.
- Acids → **coagulative necrosis** of superficial layers.
- The injured esophageal wall will be replaced by fibrous tissue → **stricture**.
- Swallowing alkaline batteries is likely to be complicated by either esophageal stricture or pyloric stricture.

- ▶ **Etiology:** accidental suicide
- ▶ **C/P:** H/O, dysphagia, burns, shock
- ▶ **Comp.:** malnutrition, dehydration, perforation, laryngeal edema, chest infection, malignancy
- ▶ **TTT:** -Emergency management
-Definitive: dilatation if failed surgery

Clinical Picture

History

- Ingestion of caustic material.

General

- Toxicity, high fever, neurogenic or hypovolemic shock.

Local

- Burns of the lips, mouth or tongue.
- Chest pain, dysphagia.

Complications

- Shock
- Malnutrition
- Dehydration
- Perforation → **mediastinitis**.
- Laryngeal edema {Early cause of death}
- Chest infection {late cause of death}
- Malignant transformation {very rare}

Treatment

Emergency Management *A B C +*

⇒ At the site of the accident:

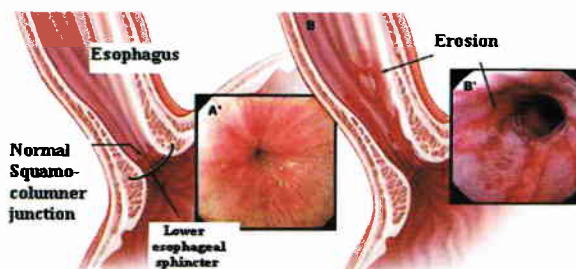
- **Patent air way** may be needed by an opening in cricothyroid membrane.
- **Ensure** good breathing.
- **Ask patient** to swallow **milk** to dilute corrosive effect.

⇒ At the hospital:

- **gastric lavage is contraindicated**
- **Antishock** measures & sedation.
- **Antibiotics** (Penicillin & Flagyl).
- **No oral intake** for 1-2 weeks.
- **Steroids** (to ↓ stricture formation & laryngeal edema)
- **Plain X- Ray** abdomen & chest to detect perforation.

Gastrografin is essential

- Before endoscopy to diagnose the extent of stricture and cases with double level stricture.
- This is a preparatory step before dilatation.



(A) normal esoph. compared with corrosive esoph. (B)
Endoscopic views

Esophagoscopy:

- Esophagoscopy is performed after 24hrs except in cases with laryngeal edema or in patients in whom the perforation is suspected
- The aim of the esophagoscopy is to confirm the presence of the corrosive injury

Definitive treatment:

A- Repeated dilatation: if fails → surgery

B- Surgery:

⇒ **Indications:**

- Complete stenosis.
- Failure of dilatation.
- Tracheo-broncho-esophageal fistula.
- Complications.

⇒ **Type of surgery:**

We do gastrostomy as a minor procedure to improve the patient condition before the definitive surgery.

– **Transhiatal blunt esophagectomy + esophageal replacement by:**

- Colon replacement or bypass.
- Jejunal loop.
- Stomach pull up.

- ▶ Replacement is preferred because the strictured esophagus can be precancerous
- ▶ Bypass is done when replacement can't be done.

IV- Esophageal FB

Site of Impaction

- At upper end (**COMMONEST**).
- At sites of normal constrictions.
- Sharp objects: anywhere.

If the foreign body is radio opaque it will be visualized by a plain x-ray, otherwise barium swallow will reveal it.

Treatment

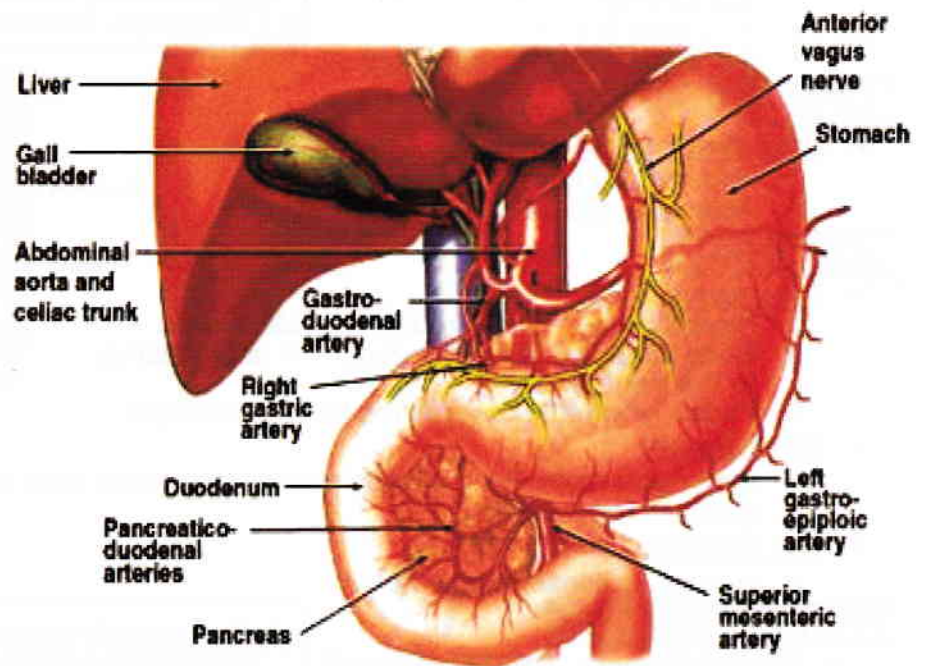
- F.B. can be removed by the rigid or fiberoptic endoscope

Therapeutic uses of esophago-gastroduodenoscopy

1. Band ligation or sclerotherapy of varices.
2. Control of bleeding from peptic ulcer by submucous adrenaline injection, laser or hemo-clips. Bleeding from superficial gastric erosions can be controlled by argon beam coagulator.
3. Percutaneous endoscopic gastrostomy (PEG).
4. Dilatation of benign strictures of the esophagus and achlasia.
5. Stenting of malignant strictures of the esophagus.
6. recently areas of esophageal dysplasia or Barrett's esophagus can be ablated superficially by endoscopic mucosal resection (EMR) or photodynamic therapy.
7. Removal of swallowed F.B.
8. ERCP with removal of CBD stones.

CHAPTER 3

Anatomy of the Stomach



Anterior View

THE STOMACH

Congenital Hypertrophic Pyloric Stenosis (CHPS)

Definition

- Thickening of the pylorus more than 8 mm. (hypertrophied)
- Normal thickness: 4 mm.

Incidence

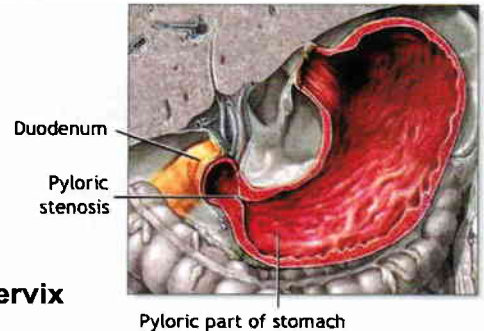
- 0.4%
- Familial (especially if the mother had CHPS)
- ♂: ♀ ratio is 4 : 1.
- It is common in the first infant.

Etiology

- Unknown, may be due to:
 - Hypertrophy of pyloric muscle
 - Achalasia: failure of pylorus to relax

Pathology

- The pylorus:
 - Hypertrophy** in the **circular muscle fibers**.
 - Ends abruptly** at the duodenum → felt as **cervix in the vagina**.
 - Fades gradually** towards the body of stomach.
- The stomach:
 - First **hypertrophied** to overcome the obstruction, later on → **dilated** with gastritis from retention.
- The intestine:
 - Normal, collapsed and empty.



Clinical Picture

- ⇒ **From 2-6 weeks, NEVER at birth** (peak at 3rd → 4th weeks and rarely present after 3 months of age).
- ⇒ Usually 1st born infant.

➤ Any neonate presenting with **projectile, non bile stained vomiting** should be considered to have CHPS until proved otherwise

Symptoms

- ⇒ One month after birth, the **mother** complains that her infants has:
 - Projectile vomiting**:
 - It is **NOT BILE STAINED** (only milk), does not respond to anti-emetics, during or shortly after meals
 - After which hunger pain occurs.
 - Constipation**: dry firm stools.
 - Failure to thrive**: progressive weight loss and dehydration.

Signs

- **General**:
 - Weight loss**.
 - Dehydration**: (Sunken eyes - depressed fontanelles – dry tongue – inelastic skin – oliguria)
 - Bad chest** (aspiration).
- **Local**: *After a feed, in a good light: (Test Feed)*
 - Upper abdominal distension.
 - Visible peristalsis**: from left to right.
 - Olive like** lump is felt in Rt. hypochondrium (**Tumor's sign**) → better to be palpated during nursing

Complications

1. Dehydration.
2. **Metabolic alkalosis** (hypochloremic alkalosis) due to HCl loss by repeated vomiting.
3. $\downarrow$ Ionized $\text{Ca}^{+2} \rightarrow$ tetany.
4. **Paradoxical aciduria** (kidneys will excrete H^+ ions instead of Na^+ , K^+)
5. Chest infection & aspiration pneumonia.
6. Liver impairment in 2% (due to bad nutritional status).

DD Of neonatal vomiting

1. **Gastroenteritis**
 - Fever, vomiting, diarrhea and loss of appetite.
2. **Pylorospasm:**
 - No palpable mass, responds well to antispasmodics.
3. **I.O.**
4. **Intracranial hemorrhage**
 - History of trauma.
 - Scalp hematoma.
5. **Feeding problems**
6. **Duodenal atresia**
 - **C/P:** soon after birth
 - Bile stained vomitus
 - **Etiology:** - True : failure of canalization
- False: annular pancreas
 - **Inv:** x-ray : Double bubble sign
 - **TTT:** R&M then duodenojejunostomy



Investigations

1. For diagnosis:

1. **U/S: (most diagnostic)**
 - Thickening of the pyloric muscle + dilated stomach.
 - (At first, we fill the stomach with saline).
2. **Gastrographin study:**
(Barium is better to be avoided in infants $\rightarrow$ lupoid pneumonitis)
 - Dilated stomach.
 - Delayed emptying.
 - Persistent narrow pyloric canal (**string sign**).

2. For complications:

1. **Chest X-Ray:** chest infection
2. **CBC:** $\rightarrow$ anemia.
3. **KFTs:** $\rightarrow$ prerenal failure.
4. **Serum electrolytes** $\rightarrow$ $\downarrow$ Na^+ , K^+ & Cl^+ & paradoxical aciduria.
5. $\downarrow$ HCl with $\uparrow$ total acidity.

Treatment

► Surgical Treatment

$\Rightarrow$ **Preoperative preparation:** Correct general condition

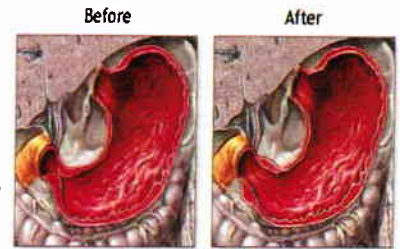
Usually it takes 24 hours to rehydrate the baby

1. **Rehydration by NaCl** & when urine becomes normal give KCl.
2. TPN & Nasogastric wash of stomach by saline.



⇒ **Ramstedt's Pyloromyotomy** [□]:

1. Transverse incision (Gridiron incision) in the Rt. hypochondrium.
2. Pylorus is grasped between index and thumb fingers.
3. Incision in the **anterosuperior aspect** of the pylorus (as it is the least vascular).
4. The serosa & musculosa is cut **till the mucosa bulges**
5. Squeeze the stomach to ensure intact mucosa.
6. If mucosal injury → repair with interrupted sutures, and reinforced by omental patch.



Peptic Ulcer

Definition

- It is ulceration of mucosa bathed in gastric juice. (Extending to muscularis mucosa)

Sites

1. **Duodenum:**
 - COMMONEST
2. **Stomach:** Less common.
3. **Rarely in:**
 - Jejunum → Zollinger Ellison \$ or after gastrojejunostomy.
 - Esophagus → reflux esophagitis.
 - Meckle's diverticulum → ectopic gastric mucosa.

Acute Peptic Ulcer (Acute Erosive Gastritis - Acute Hemorrhagic Gastritis)

Causes

- ▶ **Etiology:** NSAIDs, stress, irritants
- ▶ **C/P:** H/O of the cause, hematemesis, melena, epigastric pain & tenderness
- ▶ **Invest.:** urgent endoscopy
- ▶ **TTT:** mainly conservative, endoscopy, surgery

Types	Multiple Erosions	True Stress Ulcer
Causes	Non-steroidal	• ICU patients • Severe trauma • Major burns • Endotoxic Shock
Pathology	• Site: body & fundus of stomach • Multiple, shallow & punched out • They varies in size from 1 mm to 1 cm or more • Usually limited to the mucosa and submucosa	Multiple erosions that if not recognized and treated → coalesce to become → acute hemorrhagic gastritis

- ➡ In burns both types of ulcer occur.
- ➡ True stress ulcers (curling's ulcer) occur early in the body or fundus of stomach while an acute duodenal ulcer occurs late during convalescence.

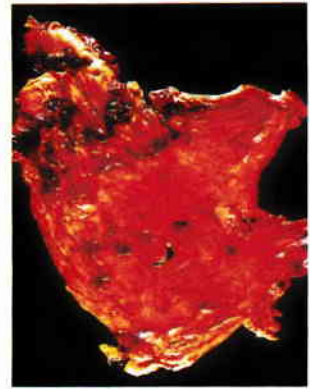
Clinical Picture

⇒ History of the Cause:

- Then the patient usually presents with hematemesis and/or melena, with epigastric pain.

⇒ Local Examination:

- Tenderness in the epigastrium.



Complications

1. Hematemesis & melena.
2. Chronicity (rare).
3. Perforation (Very rare).

Investigations

⇒ Urgent Fiberoptic Endoscopy:

- Visualize ulcers & the congested mucosa.

Ba meal is contraindicated

Treatment

Conservative (mainly)

A- Resuscitation

1. Ryle tube:

- Aspiration of fluid.
- Gastric lavage wash by cold saline & antacids (hemostatic → Vc) or sucralfate or prostaglandins.

2. IV line:

- Blood transfusion if needed.
- Sedatives.
- **Cimetidine**. If failed; IV Vasopressin.

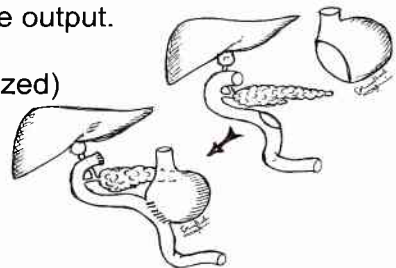
3. Catheter: (for follow up of urine output for fear of ARF).

B- Monitoring:

- Pulse.
- Temperature.
- BP.
- Urine output.

Endoscopy

1. Local injection of vasoconstrictive agents. (if localized)
2. Laser photocoagulation. (if localized)
3. Ice-cold saline wash mixed with adrenaline.



Surgical

A- Indication:

1. Failure of medical treatment. (very rare)
2. Complicated with perforation.

B- Methods:

1. Generous gastrectomy.

2. Leave a small cuff of fundus to anastomose with the intestine.

Generous Gastrectomy

- Respiratory failure and coagulopathy has been shown to increase significantly the incidence of GI bleeding from stress gastritis in ICU patients.

- Measures that are effective in preventing stress gastritis bleeding in critically ill patients:

1. Correcting any shock like state to improve systemic circulation.
2. Correcting systemic acid-base abnormality.
3. Maintain adequate nutrition.
4. Reducing intragastric acidity by either acid titration or H₂ blockers.

Chronic Peptic Ulcer

Definition

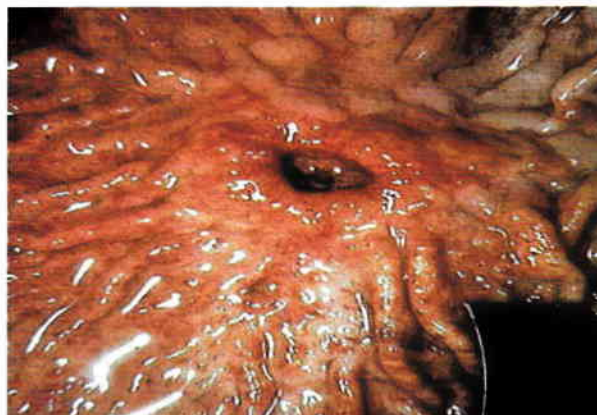
- It is ulceration of mucosa & erosion of the muscle coat.
- DU:GU → 25:1 in Egypt (while in developed countries it is 4 : 1)

Etiology

- **Etiology:** DU (↑acidity), GU (↓mucosa)
- **C/P:** pain, hematemesis, melena, pointing sign
- **Comp.:** perforation, fibrosis, bleeding, malign.
- **Invest.:** endoscopy, Ba meal, cause, comp.
- **TTT:** - **Medical:** lifestyle + drugs
- **Surgical:** DU: vagotomy ± antrectomy
GU: partial gastrectomy

Due to disturbance of balance between gastric acidity & gastric mucosal resistance

Hyperacidity (Mainly in DU)	Devitalization of mucosa (Mainly in GU)
1. Infection (by <i>Helicobacter pylori</i>). Found in mucosa of 85% of cases.	1. Infection (by <i>Helicobacter pylori</i>). Found in mucosa of 75% of cases.
2. Irritant foods smoking, alcohol, drugs as NSAIDs	2. Deficient mucous barrier as atrophic gastritis (most common cause)
3. Liver diseases (lack of inactivation of histamine & gastrin)	3. Irritant foods smoking, alcohol, drugs as NSAID
4. Endocrinal disturbances as in ZES (gastrinoma) & hyperparathyroidism	4. Pyloroduodenal reflux of bile (biliary ulcer worsens with antacids) → back diffusion of H ⁺ → mucosal damage
5. Genetic factors: <u>blood group O</u> ☐	5. Antral stasis
6. Increased vagal tone as in nervousness & stress	6. Vitamin A & C deficiency (decrease regeneration of cells)
7. Stasis of food (leads to hypergastrinemia)	7. Ischaemia (small minute embolus → small ulcer → HCl continues to erode
▪ In Gastric ulcer → usually associated with hypoacidity. ▪ In duodenal ulcer → usually associated with hyperacidity.	



Pathology

		DU	GU
Gross picture	Number	Single (usually kissing)	Single
	Site	1 st inch of 1 st part of duodenum (duodenal cap) anterior or posterior or both (kissing ulcer)	Lesser curvature prepyloric (the ulcer bearing area)
	Size	Usually small	Larger than DU
	Shape	rounded or oval	the same or saddle
	Edge	Sloping (at first) or punched out (later)	
	Margin	Congested with mucosal folds	
	Floor	1. Penetrate the muscle coat 2. Filled with: - Granulation tissue during activity - Fibrous tissue during healing	
	Base	Indurated (due to fibrosis) with destruction of mucosal coat.	
Microscopic	Base	Dense fibrous tissue PNL, lymphocytic infiltration, nerve ending around ulcer are thickened (bulbous)	
	Arteries	endarteritis obliterans	

- ▶ Post. wall DU ulcer is more dangerous → penetrates pancreas → chronic pancreatitis & penetrate gastroduodenal artery → severe hematemesis.
- ▶ GU:
 - Type I → in body, on the lesser curve associate with decrease HCl & atrophic gastritis (60%)
 - Type II → GU + DU (20%); DU develops first then causes GU due to gastric stasis.
 - Type III → in pyloric antrum or canal (20%).
- ▶ Types II, III are associated with hyperacidity.
- ▶ How to know the site of ulcer intraoperatively?
 1. White scarring.
 2. Delicate vascular adhesions with the surroundings.
 3. Enlarged LNs.
 4. Stippling sign: rub with gauze → petechiae
 5. In doubtful cases: gastrotomy → ulcer.
- ▶ A gastric ulcer situated away from lesser curve is highly suspicious of malignancy.

Clinical Picture

(Symptoms does not allow accurate discrimination)

			DU	GU
Symptoms	Type of patient	Type	- Male 25-40 yrs usually well fed with no apparent signs of ill health - Male : female = 5 : 1	- Male 35-45 yrs usually thin and underweight - Male : female = 2 : 1
	1. Pain	Time	Burning, stabbing, colic, shooting or dull aching a. 2 – 3 hrs^{sq} after meal (due to passage of acid chyme on the ulcer after gastric evacuation) & persists till the next meal (as hunger pain) b. Nocturnal pain awakens the patient in early morning. <u>due to</u> : 1. Maximum HCl secretion at night. 2. Stomach remains empty for a long time.	Usually starts $\frac{1}{2}$ - 1 hr or immediately^{sq} after meals
		Site	Above the umbilicus to the Rt. side of the midline	Epigastric in the midline or just to the left. May radiate to the back.
		Exciting Factors	Irritant foods, nervousness, Smoking, stress	
		Relieved By	Alkalis & Antacids	
			Food or anything the buffers the gastric juice (patient carries biscuits)	Fasting & vomiting If antacids fail to stop pain → possibility of malignancy or penetration
		Periodicity	Marked by: - Weather, worry, work - It occurs for 2 - 6 wks & relieved for 2 - 6 months - It is commoner in spring & autumn	Not marked
	2. Vomiting		Not prominent (Only if pyloric stenosis occurs)	Very common as it relieves pain & may be self induced
	3. Appetite		Good as eating relieves pain except for alcohol and smoking	Good but the patient is afraid to eat (sitophobia) as it induces pain
	4. Body wt.		Gain weight	Loss of weight
	5. Hematemesis & melena		+ve (<u>more melena</u>)	+ve (<u>more Hematemesis</u>)
	1. Anemia		+ ve	+ ve
	2. Pointing sign		The patient can localize site of pain exactly by one finger	
Signs	3. Tenderness		Above the umbilicus to the Rt. side of the midline	In the epigastrium in the midline or just to the left of it.

MCQ: However hematemesis / melena are more common with DU as incidence of DU > GU

Complications

A- Perforation:

1. Acute → generalized peritonitis.
2. Subacute → localized subphrenic abscess.
3. Chronic → penetration to:
 - **Pancreas** → pain radiating to the back.
 - **Liver** → pain radiating to the shoulder.

B- Bleeding (5 %)

1. Mild → anemia (due to chronic blood loss).
2. Moderate → hematemesis, melena & shock.
3. Severe → fatal (especially if eroding great blood vessels e.g. gastroduodenal).

C- Fibrosis:

⇒ Pyloric stenosis, hourglass stomach:

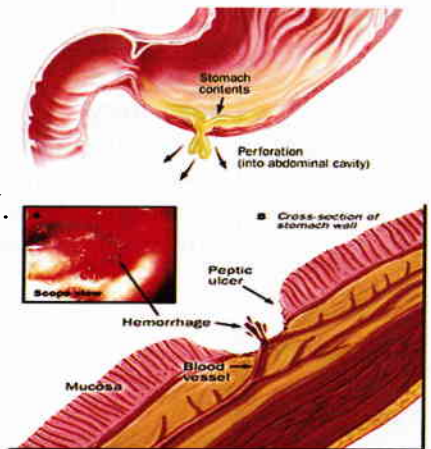
- Repeated vomiting of offensive undigested fermented food that is usually **bile-free** (associated with a tetanic spasm).

D- Malignancy:

⇒ (Only in GU) & very rare (< 1 % of cases):

- **Malignant ulcer is usually malignant from the start.**

E- Recurrence.



DD

1. Gastric Lesion:

- Acute ulcer.
- Gastritis.
- Carcinoma.
- Hiatus hernia.

2. Wilkie's Triad:

- Chronic peptic ulcer + chronic cholecystitis + chronic appendicitis.

3. Other Causes of Dyspepsia.

Investigations

A- For diagnosis:

➤ Upper GI endoscopy (investigation of choice):

1. For diagnosis.
2. For follow-up in patients under ttt (if healed → ulcer white)
3. Obtains 4 quadrant punch **biopsy** + brush cytology. (esp. in GU).
4. Detects the cause of hematemesis.

➤ **Barium meal:**

	DU	GU
Ulcer niche	In the duodenal cap	on the lesser curvature.
Deformity	<u>Early</u> due to muscle spasm <u>Later on</u> due to fibrosis (trifoliate deformity) To be sure that the deformity is due to either spasm or fibrosis & not due to peristaltic wave, Serial duodenal film must be taken	Ulcer niche with opposite notch due to spasm of circular muscles. Later on due to fibrosis of circular muscle fibers
Post-evacuation	Ulcer crater → • A flake of Barium remains in the ulcer niche (in patients with evacuation failure) • The mucosal folds are seen radiating toward it	
Tenderness	Tenderness at site of the ulcer	
Complications	Soup dish appearance (in pyloric stenosis)	Hourglass or teapot appearance

➤ **CXR:** Air under diaphragm → perforated peptic ulcer.**B- For complications:**➤ **For bleeding**➤ **CBC:**

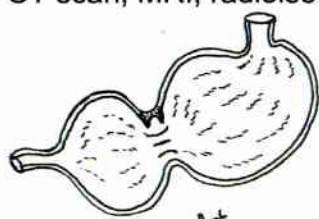
- Microcytic hypochromic anemia due to chronic blood loss.

➤ **Stool analysis:**

- By benzedine test for occult blood loss.

➤ **In recurrent cases, we should search for the cause:**

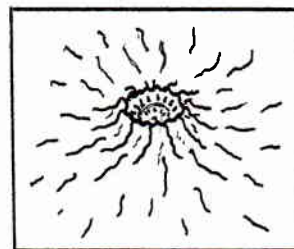
- H. Pylori → **biopsy, serology & urease test** (biopsy + urea + pH indicator → **organism + urea → NH₃ changes the color of indicator**).
- Endocrinal causes (esp. if multiple ulcers)
 - ZES → **Gastrin assay**
+ **Octreotide CT scan** to localize tumor.
 - Hyperparathyroidism → **Serum calcium, phosphorus (MEN \$)**
+ CT scan, MRI, radioisotope subtraction scan



Hourglass stomach



Ulcer niche



Ulcer crater

Treatment

Treatment of uncomplicated cases:

- A- Medical (mainly).
- B- Surgical.

Treatment of the cause:
e.g. ZES,
hyperparathyroidism*Treatment of complicated cases:*
(See later)

Medical

TTT of uncomplicated peptic ulcer is mainly medical

A- Aim:

- Creating the balance between acid secretion and mucosal barrier.

B- LIFE STYLE MODIFICATION:

1. **Rest:** Physical & mental rest (sedatives might be needed)
2. **Small frequent meals:** about 5 times a day
3. **Avoid irritant foods** → spicy etc.
4. **Avoid irritant drugs** → aspirin, cortisone etc.
5. **Avoid** → smoking, alcohol, coffee, tea and cola.

C- Treatment of H. pylori by triple therapy:

Omeprazole + Metronidazole + Clarithromycin.

D- Under these regimens:

- The ulcer heals within 6 weeks but full dose ttt is continued for few weeks more.
- Endoscopy is needed to evaluate healing.
- High rate of recurrence.

E- Disadvantages of long term therapy with omeprazole :

1. Expensive
2. Atrophic gastritis & bacterial overgrowth with omeprazole
3. Prolonged gastrinemia → Gastric neoplasia is a theoretical risk

Other drugs used in peptic ulcer:➤ **Drugs decreasing HCl secretion:**A. **H₂ blockers:**

1. Cimetidine 800mg at bedtime.
(To avoid contact of acid with empty stomach)
2. Ranitidine 150mg twice daily.
3. Famotidine 20mg twice daily.

B. **Proton pump inhibitor:**↓↓ **Gastric acidity to zero in a matter of days**

- Omeprazole 20mg in the morning.

➤ **Antacids:**

1. Mg hydroxide.
2. Mg trisilicate.
3. Ca carbonate.
4. Aluminum hydroxide.

➤ **Coating agents**

1. Sucralfate.
2. Bismuth.

➤ **Drugs enhance mucosal barrier:**

1. Carbenoxelone.
2. Prostaglandins.

- i. Medical treatment of GU is almost exactly as that of DU. However, repeated endoscopy must be carried out after 6 weeks regardless of symptomatic improvement.
- ii. If GU shows:
 - No attempt at healing: it should be considered malignant and surgery is performed
 - Partial healing: give medical treatment for another 2 months (never > 6 months)
- iii. The main disadvantage of medical treatment of DU is high risk of recurrence after stoppage of treatment; to overcome this problem:
 - Interval therapy: give full dose regimen at times of maximum stress.
 - Small dose at bedtime for prolonged period.
- iv. It is reasonable that a doctor managing a patient with uncomfortable PU should suggest modifications to the patient's life style. Particularly cessation of smoking. This advice is rarely followed and the mainstay of treatment is drugs.

The precancerous possibility of GU makes it different from DU in²:

1. Regular follow up by endoscopy is indicated.
2. Surgery if no response to medical treatment after 6 weeks.
3. Aim of surgery is to completely remove the ulcer.

Surgical

A- Indications:

1. Failure of medical treatment. (in case of gastric ulcer → failure to start healing after 6 weeks or to achieve it within 6 months)
2. Complications.
3. Incompliant patients.
4. Patient cannot pay for the drug.
5. If malignancy can't be excluded in GU.

B- Methods:

1. Duodenal ulcer:

- **Aim of surgery:** not only to heal the ulcer but also to provide adequate protection against recurrence.
- **Principles of surgery:** achieve permanent and adequate reduction of gastric acid secretion By either
 - Abolishing the nervous phase of gastric acid secretion by Vagotomy which may be :
 - Truncal
 - Selective
 - Highly selective
 - Seromyotomy
 - Abolishing the hormonal phase by antrectomy. Some surgeons combined vagotomy and antrectomy
 - Reduction of parietal cell mass by a subtotal gastrectomy → Historical

	WHAT IS CUT?	STRUCTURES THAT MAY BE INJURED ?	RESULTS	COMPLICATIONS & PROGNOSIS
Truncal Vagotomy	كل حافة Trunk of Ant. & Post Vagus	- Hepatic Br. - Celiac Br. - Crow's Foot	Hepatic Br. - Gall stones (dyskinesia of sphinct of oddi) Coeliac Br.: - Distension of Stomach - Repeated episodes of Infective Diarrhea . Crow's foot : - Loss of innervation of pylorus Others - Incomplete procedure - Recurrence	Ⓢ Pyloroplasty (mikulicz tech) Ⓢ Gastro-Jejunostomy : - Posterior, - Retrocolic, - Isoperistaltic
Selective Vagotomy	Ant. & Post. Nerve of Lateraljet	Crow's foot	Loss of innervation of pylorus	Pyloroplasty, Diarrhea, dumping
Highly selective vagotomy	Branches innervating body of Stomach stopping at Incisura (spare Br. Of pylorus) و بينفد ال Ant. & Post. Nerve of Lateraljet		Pylorus is opened, so need for pyloroplasty → so, no diarrhea	Necrosis of Lesser Curvature in 0.5 % Contra-indications : - PU + Gastric outlet \$ - PU in Fundus - Cigarette chain smoking - Perforated PU
Sero-Myotomy	نسيب عرض صباع و نقطع ال Serosa & Musculosa		No Necrosis on Lesser Curvature	Resolution

► Gastric Drainage Procedure :

- 1- Gastrojejunostomy
- 2- Pyloroplasty: has many types the most popular is the Heineke-Mikulicz type where the pylorus is divided longitudinally and sutured transversely.

► In this country the 2 most popular operations are :

- 1- T.V. & gastrojejunostomy
- 2- Highly selective vagotomy

2. Gastric ulcers:

- **Aim of surgery:** Remove the ulcer or the whole entire ulcer bearing area & to ensure proper gastric emptying.
- **Operation :**
 - Partial gastrectomy is the standard procedure , it removes the ulcer together with the entire ulcer bearing area
 - The operation is ended by gastroduodenal anastomosis (billroth1) or less common gastrojejunostomy (billroth 2)

Complications of PU

A- Acute Complications:

- ⇒ That occur during periods of ulcer activity:
1. Perforation.
 2. Bleeding.

B- Chronic complications:

- ⇒ That occur insidiously:
1. Fibrous contracture of ulcer produces pyloric stenosis or hourglass stomach.
 2. Penetration.
 3. Malignant transformation of GU.

1 - Perforation

Acute Perforation

Incidence

- More common in **DU (90%)**
- More common in **males**.
- More common in **anterior wall ulcers**.

Predisposing Factors

- All factors which cause exacerbation as stress, NSAIDs or heavy meal.
- It might be silent until perforation occurs.

Stages

A- Stage of Chemical Peritonitis:

Pathology

- There is a sudden rupture of the ulcer base → gastric or duodenal contents in peritoneal cavity → chemical peritonitis.

- **Etiology:** stress, NSAIDs, heavy meal
- **C/P:** ♂ with H/O of DU suddenly develops acute abdomen in 3 stages: ①Chemical peritonitis ②Quiescent st. ③Septic peritonitis
- **Invest.:** X-ray abdomen Erect, U/S, CT, aspiration test, for comp.
- **TTT:** R&M + urgent operation:
 - **Bad:** omental patch
 - **Good:** definitive surgery

Clinical Picture

➔ Symptoms (usually short and the patient not seen in it)

- Sudden severe pain in epigastrium, which then becomes generalized.

➔ Signs

➤ General:

- Pallor, sweating, subnormal temperature, rapid weak pulse.

➤ Local:

- **Inspection:** Board like rigidity,
- **Palpation:** guarding, epigastric tenderness .
- **Percussion:** ↓ liver dullness (air under the diaphragm).
Shifting dullness (fluid in the peritoneal cavity).
- **Auscultation:** ↓ intestinal sounds (Paralytic ileus occurs late).
- **P/R:** fullness in Douglas pouch in females & rectovesical pouch in males.

B- Quiescent Stage (Stage of Illusion):

Pathology

- It occurs 3-6 hours after the perforation.
- Reaction of the peritoneum against chemical agent → production of large amount of alkaline fluid and bringing antibodies.

Clinical Picture

➔ Symptoms

- Pain decreases.

➔ Signs

➤ General:

- The patient has more tachycardia.

➤ Local:

- Like the previous stage with less rigidity and ↑ shifting dullness.

C- Stage of Septic Peritonitis:

Pathology

- It occurs 6-12 hours after the perforation.
- Bacteria (flourish as a result of decreased HCl and loss of its antiseptic effect) → pus formation → septic peritonitis.

Clinical Picture

➔ Symptoms

- Pain increases with fever, anorexia, headache, malaise, repeated vomiting & distension (due to paralytic ileus).

➔ Signs

➤ General:

- Fever, toxemia, deterioration of the general condition of the patient, more tachycardia

➤ Local:

- Generalized rigidity, tenderness, progressive abdominal distension.

- ▶ DU is less serious than gastric ulcer because of its smaller, less irritant and more sterile contents. Poured out contents flow along paracolic gutter to the caecum while gastric ulcer perforates directly into peritoneal cavity.
- ▶ The fluid from perforated DU might run on the paracolic gutter giving picture simulating **appendicitis**.
- ▶ During appendectomy, if there is bile in peritoneum → put a drain in the wound of the appendectomy + exploratory midline laparotomy to deal with the ulcer

DD

a. From causes of upper abdominal pain:

1. Acute cholecystitis.
2. Acute pancreatitis.
3. Mesenteric vascular occlusion (MVO)
4. Dissecting aortic aneurysm.
5. Inferior wall myocardial infarction.
6. DKA.

b. From acute appendicitis:

- If it presents by Rt. iliac fossa pain.

Investigations (URGENT)

1. Radiological:

- **Plain X-Ray abdomen erect** → air under the diaphragm (absence of air under diaphragm doesn't exclude perforation).
- **U/S:** shows fluid in peritoneum.
- **Gastrografen meal:** ensure escape of the dye through the perforation (especially if no air under diaphragm is seen by X-ray)
- **CT** (more accurate)

- ▶ **Ba meal** → chemical peritonitis
- ▶ **Endoscope** → difficult to diagnose

2. Instrumental:

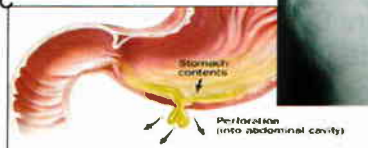
- **Aspiration test:** (guided by U/S)
 - Bile stained alkaline fluid from the peritoneum.
 - Might show pus in case of septic shock.

3. Laboratory:

- **CBC:** ↑ PNL in septic peritonitis.
- **KFTs:** prerenal failure.
- **Electrolytes:** disturbance.
- **Serum amylase:** moderate increase (800 somogyi units)

Perforation

- Patient presented with acute abdominal pain
- Plain X-ray chest & abdomen showed air under the diaphragm



Treatment

(Urgent operation after good preparation)

1. Resuscitation & Monitoring:

a- Resuscitation:

- **Ryle:** continuous aspiration.
- **IV** (fluids, blood, antibiotics, analgesics, H₂ blockers, omeprazole).
- **Catheter**

b- Monitoring: of BP, pulse, urine output, CVP, electrolytes and pH.

2. Emergency Operation: (the simplest procedure is done)

⇒ *Peritoneal toilet then according to condition of the patient:*

Bad general condition or late presentation (> 12 hr):

- 1- Simple closure of the perforation with **omental patch** (Graham's method) with postoperative antiulcer TTT.
- 2- However, in GU, biopsy must be taken.

Good general condition or early presentation (< 12 hr):

Definitive Surgery

1. DU → Vagotomy & drainage.
2. GU → partial gastrectomy.

⇒ *Then draining of the peritoneum.*

3. Postoperative Care:

- Medical treatment of the ulcer for patients who do not undergo definitive surgery.

Subacute Perforated Peptic Ulcer

- This is a small perforation allowing only a minimal amount of contents to enter the peritoneal cavity and is rapidly sealed
- It gives the same early signs as acute perforated PU but much reduced.
- If diagnosed correctly it can be treated conservatively

Chronic Perforation of Peptic Ulcer (Penetrating Peptic Ulcer)

- Means erosion in an adjacent structure as the pancreas.
- It is suspected when the pain becomes more or less persistent and referred to the back

2-Bleeding Peptic Ulcer

Incidence

- One of the most common causes of hematemesis.
- About 20% of patients (especially elderly) with ulcer disease would suffer from bleeding episodes in the form of hematemesis or melena.

- ▶ **Etiology:** as perforation
- ▶ **C/P:** ♂ with H/O of PU develops hematemesis, melena, BPR, anemia, shock
- ▶ **Invest.:** urgent endoscopy, angiography
- ▶ **TTT:** R&M + endoscopy ± surgery

PDF

1. Irritant food.
2. Ulcerogenic drugs.
3. Stress.



The ulcer is deep, with sharp proximal edge & a sloping distal edge
The arrow points to an eroded gastric artery which has caused fatal hemorrhage

Pathology

➤ Hematemesis:

1. On microscopic level as a part of normal disease process
2. **Mild** → Bleeding from the granulation tissue.
3. **Moderate** → Due to erosion of small vessels.
4. **Severe** → Due to erosion of a large vessel as **gastroduodenal artery**☐.

Clinical Picture

Symptoms

➤ Past history of Ulcer dyspepsia may be present then:

1. **Hematemesis** vomiting of coffee ground blood due to acid hematin (may be fresh blood in severe cases due to upper GIT lesion proximal to ligament of Treitz).
2. **Melena** (passage of soft black tarry offensive stool) in mild cases.
3. **Fresh blood per rectum** in severe cases.

Signs

➤ General:

1. Anemia in case of repeated minor bleeding.
2. Hypovolemic shock (sweating, pallor, weak rapid pulse).

➤ Local:

- Epigastric tenderness.

DD

From other causes of hematemesis

Complications

1. Severe shock .
2. Renal failure.
3. Respiratory complication.

Investigations

(Urgent investigations after resuscitation)

1. Upper GI endoscopy
 - For diagnosis and exclusion of other causes of hematemesis.
2. Selective mesenteric angiography
 - If endoscope fails to localize the source.
3. Laboratory:
 - CBC
 - Electrolytes.
 - KFTs.

Treatment

(Most of cases stop bleeding spontaneously due to clot formation at base of ulcer)

1. **Correction of general condition of the patient (Resuscitation + Monitoring)**
 - Bed rest.
 - Antishock measures (blood, IV fluids, warmth, O₂)
 - Monitoring → urine output & better by CVP.

2. Once the patient is stabilized → urgent endoscopy

Diagnostic

Exclude other causes of hematemesis

Therapeutic

Laser coagulation, thermal coagulation, injection by alcohol or adrenaline in ulcer base.

3. IV H_2 blocker or omeprazole.

4. Antacids.

5. NG lavage by cold saline.

Indications for Endoscopic treatment:

- 1- The ulcer is seen to be actively bleeding
- 2- Visible vessels in the base
- 3- Fresh clot covering the ulcer

■ The following cases usually respond well to conservative treatment:

- 1) Young patients under 45 years old.
- 2) Minor bleeds.
- 3) Short history of dyspepsia.
- 4) Recent history of ulcerogenic drug intake.

➡ Surgical Treatment:

- ▶ The decision of surgery should be taken early within 48-72 hrs as the results of late surgery are poor.

➤ Indications:

A. Absolute Indications:

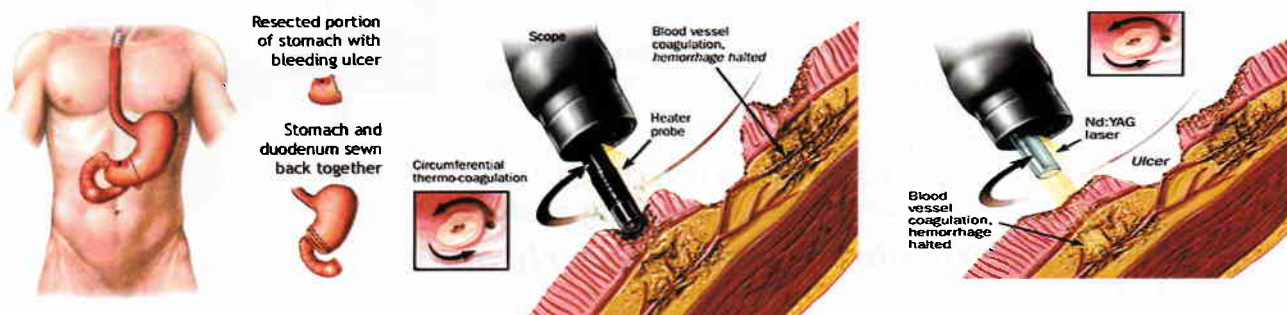
1. Failure of conservative treatment.
2. Severe bleeding from the start of about 2L or more (> 4 units of blood needed for correction).
3. Continuous bleeding.(as evidenced by the need to transfuse 1000 ml of blood / day)
4. If bleeding recurs while patient is in the hospital

B. Relative Indications:

1. Old patient due to atherosclerosis.
2. Associated pathology as perforation.
3. Long history of ulcer disease

➤ Methods:

- If GU → partial gastrectomy.
- If DU → Truncal vagotomy + pyloroplasty + under running sutures for hemostasis. Gastroduodenal artery may require ligation.



3- Fibrous Contracture

Pyloric Stenosis

Definition It is actually a duodenal stenosis

- Narrowing of pyloric canal interfering with the downward passage of gastric contents to the duodenum.

Cause

- Healing of DU → fibrosis → stenosis → post pyloric stenosis.

Pathology

1. **Pylorus** → stenosed (spasm then fibrosis).
2. **Stomach** → hypertrophic dilatation with gastritis due to stasis.
3. **Intestine** → normal.

Clinical Picture

➡ Symptoms:

a. Pain:

- History of periodic pain, which is lost at presentation becoming continuous with no relation to food.

b. Vomiting:

- Projectile, **contains food of previous day, not bile stained**, foul odour from fermentation, characteristically in the evening

c. Progressive weight loss & constipation.

➡ Signs: (We have to exclude malignancy)

➤ General:

1. Dehydration
2. Chest infection
3. Weight loss

▶ **Patient with past history of PU on medical TTT, now pain persistent and vomiting by the end of the day**

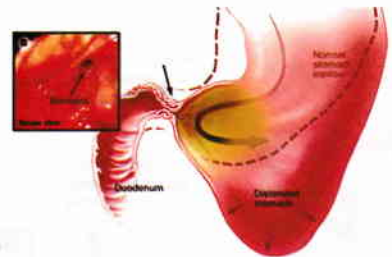
➤ Abdominal:

▪ Inspection:

1. Epigastric fullness
2. Peristaltic waves from left to right.
3. Outlines of enlarged stomach may be seen

▪ Auscultation:

- Succussion splash (the stomach full with water & solid) **(never to be done)**



DD

Of pyloric obstruction in elderly male

1. Cancer stomach.
2. TB or Crohn's disease.
3. Rarely compression of stomach by lymphoma or cancer pancreas.

Complications

(see CHPS)

1. Hypochloremic hypokalemic metabolic alkalosis → tetany.
2. ↓ Na⁺, K⁺, Cl⁻.
3. Paradoxical aciduria (see before).
4. Dehydration.
5. Respiratory tract infection.

► Signs suggestive of malignancy:

- Virchow's LN.
- Umbilical nodules
- Malignant ascites.

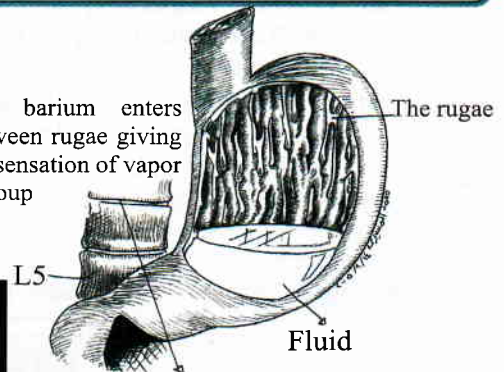
Investigations

1. Radiological:

▪ Barium meal:

- a. Dilated stomach (often reaching the pelvis).
- b. Soup Dish appearance or inverted hat appearance (Fluid level + rugae)
- c. Delayed gastric emptying (Film taken 12-24 hrs later)
- d. It may miss small pyloric carcinoma

The barium enters between rugae giving the sensation of vapor of soup



2. Instrumental:

▪ Upper GI endoscopy:

- Main value: to exclude malignancy ± biopsy
- Dilated stomach with atrophic gastritis & failure of passage through pylorus.

► Endoscopy can only be done on empty stomach and so should follow a period of fasting followed by gastric lavage.

3. Laboratory:

(assess general condition of the patient)

- CBC → anemia, hemoconcentration.
- KFTs → prerenal failure.
- Serum electrolytes → ↓ Na⁺, K⁺ & Cl⁻ & paradoxical aciduria.

Treatment (Only surgical)

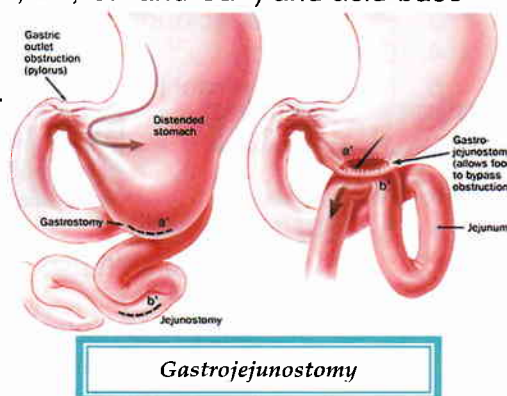
Preoperative

- a) **Correction of the general condition** of the patient.
 1. High protein fluid diet.
 2. Correction of fluids, electrolytes (Na^+ , K^+ , Cl^- and Ca^+) and acid-base balance (alkalosis)
 3. Blood transfusion for anemia.
 4. Chest physiotherapy and antibiotics.

- b) **Gastric lavage through Ryle's tube.**

Operative

- a) **Truncal vagotomy & gastrojejunostomy is the standard treatment**
- b) **In the elderly or unfit patients:**
Gastrojejunostomy alone or endoscopic balloon dilatation
- c) **Partial gastrectomy.**



- Some cases of pyloric stenosis occur due to muscle spasm around active ulcer.
- These cases respond well to medical treatment

Hourglass Stomach

- Due to cicatrized saddle shaped ulcer on the lesser Curve
- It presents by symptoms of gastric obstruction but there will be early satiety & loss of weight
- Endoscopy and barium meal will diagnose it
- TTT is by Partial gastrectomy

4- Malignant Changes in GU

Clinical Picture

- **Pain becomes constant & not relieved by treatment or vomiting** together with other manifestations of gastric cancer (see later).

Investigations

- 1- **Barium meal:**
 - Ulcer is > 2 cm & in unusual site (as greater curvature).
- 2- **Upper GI endoscope & 4- quadrant biopsy:**
 - + Other investigations of gastric cancer (see later)



Treatment

- As gastric cancer.

5-Recurrent Peptic Ulcer 3%

Etiology

- ▶ **Etiology:** persistent PDFs (↑acidity, drugs), inadequate surgery
- ▶ **C/P:** as PU + recurrent dyspepsia
- ▶ **Invest.:** for cause + as ulcer
- ▶ **TTT:** TTT of the cause + as ulcer

Persistent Hyperacidity	Inadequate Surgical Procedures	Use of Ulcerogenic Drugs
1. Zollinger Ellison \$ 2. Hyperparathyroidism	1. Incomplete vagotomy or criminal nerve of Grassi. 2. Inadequate gastrectomy → leaving part of antrum 3. Inadequate drainage (no pyloroplasty or small gastrojejunostomy).	NSAIDs Cortisone

Sites

1. Gastric.
2. Stoma.
3. Jejunal.
4. Recurrence of the same ulcer.

Clinical Picture

- Recurrent dyspepsia + other symptoms as peptic ulcer.

Investigations

(Must search for the cause of recurrence)

1. Laboratory:

1. Gastrin assay (ZES).
2. Ca & P.
3. Tests of *H. pylori*.
4. Gastric function test (for adequacy of vagotomy)

2. Radiology: Ba meal → diagnoses recurrent ulcer.

3. Instrumental: Endoscopy → diagnoses recurrent ulcer.

Treatment

Treatment of the cause

1. Stop ulcerogenic drugs.
2. Zollinger Ellison Syndrome:
 - i. Detectable tumor → Resect it.
 - ii. Undetectable tumor → total gastrectomy + distal pancreatectomy omeprazole large dose.
3. Hyperparathyroidism:
 - If adenoma → Resect it.
 - If hyperplasia → remove 3 ½ glands & implant ½ in deltoid m.

Treatment of the Ulcer

➤ Medical:

- (As peptic ulcer).

➤ Surgical:

1. If medical treatment fails.
2. If after vagotomy → antrectomy
3. If after gastrectomy → higher gastrectomy + vagotomy

Complications of Partial Gastrectomy

A- Operative Complications:

1. Complications of anesthesia.
2. Primary hemorrhage.
3. Injury of important structures.
 - Pancreas → pancreatitis.
 - CBD → obstructive jaundice, fistula
 - Spleen → bleeding.
 - Middle colic vessels → colon gangrene
4. Infection

B- Postoperative Complications:

1. Early Complications:

1. Hemorrhage:

⇒ Inside the stomach:

▪ Causes:

- Suture line.
- Missed ulcer.

▪ Diagnosis:

- Bleeding from the nasogastric tube.

⇒ Outside the stomach:

▪ Causes:

- Slipped ligature
- Torn splenic capsule.

▪ Diagnosis: Bleeding from the drain.

▪ Treatment:

- Blood transfusion + Ryle
- If failed → re-exploration.

2. Stomal Obstruction due to:

- ⇒ Edema (treated by aspiration and IV therapy).
- ⇒ Technical error.
- ⇒ Retrograde jejuno gastric intussusception:
 - Reduce spontaneously or after barium meal

3. Leakage from the anastomosis:

▪ Causes:

- Acute gastric dilatation.
- Technical fault.

▪ Pathology and treatment:

- Peritonitis (if before 48 hours):
 - Resuscitation & Monitoring.
 - Immediate laparotomy.
 - Peritoneal toilet.
 - Closure (if possible).
- Subphrenic abscess (after 48 hours):
 - Percutaneous aspiration.
 - If failed → open drainage.

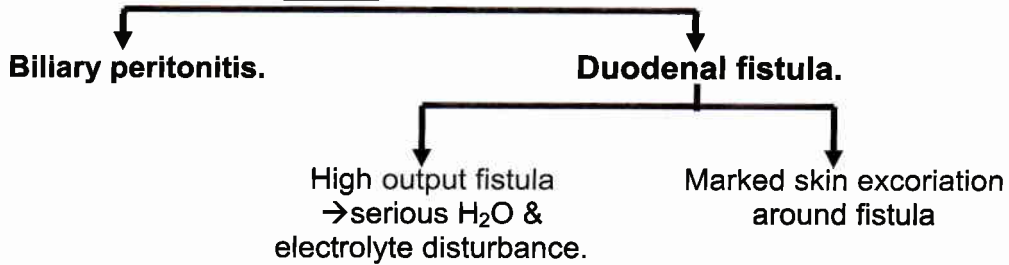
4. Duodenal Stump Blowout:

- Serious complication but infrequent.
- Responsible of 60% of mortality after gastrectomy.
- Timing: 4th day.

- **Causes:**

- Sutures taken in inflamed or scarred duodenum.
- Afferent loop obstruction.

- **Effect:**



- **TTT:**

- Sump drain + skin protection + correct H₂O & electrolyte disturbances, TPN.
- It will close so long afferent loop is patent.
- Prophylactic: Adequate closure +10-days tube drain.

5. Paralytic Ileus:

- Due to manipulation of intestine.

6. Acute Gastric Dilatation,

- From handling of stomach.

- **TTT:**

(Of ileus and acute gastric dilatation)

- Prophylactic:

- Preoperative: good fluid and electrolyte balance.
- Operative: gentle manipulation.
- Postoperative: Ryle till ileus is over.

- Curative:

- Ryle, correct fluid & electrolytes.

7. Burst Abdomen: (see Hernia).

8. Pulmonary Complications:

- Aspiration pneumonia, lung abscess, collapse, ARDS

9. Subphrenic Collection:

- **Causes:**

1. Leakage.
2. Duodenal stump blowout.
3. Hemorrhage.

- **TTT:**

- Percutaneous guided (U/S, CT) aspiration
- Operative drainage.

2. Delayed Complications:

1. Postgastrectomy Syndromes:

1. Nutritional Syndrome:

1- Weight loss.

2- Anemia: due to

- ↓ Fe due to ↓ HCl.
- Vitamin B12 deficiency due to ↓ intrinsic factor.

3- Steatorrhea:

- (↓ Acidity → ↓ pancreatic stimulation especially in Polya due to bypassing of the duodenum)

4- Calcium Deficiency:

- Duodenum is the major site of calcium absorption.

2. Afferent Loop Syndrome:

1. **Cause:** stomal edema, kink of afferent loop, narrowing by sutures of gastrojejunostomy, retrograde intussusception.
2. **C/P:** acute epigastric pain and fullness after meal followed by projectile bilious vomiting which contains no food.
3. **Mechanism:** the food has stimulated pancreatic and bile secretion which distends the afferent loop, pressure increases until obstruction is overcome and the loop empties itself into the stomach
4. **TTT:** surgical conversion of anastomosis to Roux-en-Y loop.

3. Postcibal Syndrome (Dumping Syndrome)

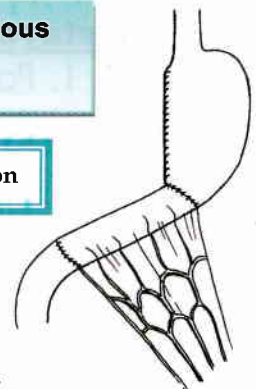
Postcibal Syndrome (Dumping Syndrome)	
Early Dumping	Late Dumping
1- Hypovolemia 2- Within ½ hour after meal 3-Due to: Loss of the reservoir function of the stomach → rapid passage of hypertonic chyme into small intestine → shift of extracellular fluid into lumen of intestine → depletion of blood volume. 4- C/P : A- <u>Gastrointestinal:</u> ▪ Abdominal fullness followed by colic and diarrhea B- <u>Vasomotor:</u> ▪ Sense of weakness, flushing, palpitation ▪ ↑ pulse, ↓ blood pressure. 5- Prevention ▪ Small frequent meals, rich in proteins and fats and less in CHO ▪ Liquids allowed only in between meals 6- Treatment: ▪ Assuming the supine position ▪ Polya converted to Roux-en-Y to delay emptying	1- Hypoglycemia 2 - 3 hours after meal ↑ CHO diet → hyperglycemia → ↑ insulin output → hypoglycemia A- Sweating. B- Hunger. C- Tremors. D- Nausea. ▪ Small frequent meals , low CHO diet ▪ Oral hypoglycemic (Tolbutamide 500mg before meals) ▪ Avoid high CHO diet ▪ Somatostatin

- In refractory cases, we decrease the rate of gastric emptying in various ways such as interposition of a reversed jejunal loop between the stomach and the small intestine.

Jejunal Interposition

2. Recurrent Ulcer (3% after 2 years):

- At the stomach remnant.
- At line of anastomosis.
- At the efferent loop.



3. Gastro-jejuno-colic fistula:

- **Pathogenesis:**

- Stomal gastrojejunal ulcer → erodes the transverse colon.

- **Clinical picture:**

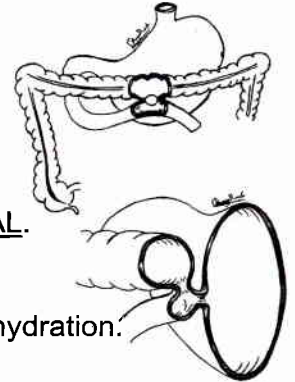
- Fecal vomiting.
- Severe diarrhea due to enteritis.
- Dehydration.

- **Investigations:**

- **Barium enema** (the best) NOT MEAL.
- Upper GI endoscopy.

- **Treatment:**

1. Resuscitation and monitoring for dehydration.
2. Proximal colostomy
3. When general condition improves:
 - Treatment of fistula by separation of colon & closure.
 - Revise the gastrectomy at higher level.
4. Closure of the colostomy after 3 months.



Gastro-jejuno-colic fistula

4. Intestinal Obstruction:

- **Jejunogastric intussusception:**

- The jejunum is telescoped into the stomach.

- **Internal hernia:**

- Through an opening in the transverse mesocolon (following gastrojejunostomy).

- **Adhesions & Fibrosis**

- **Treatment:**

- Operative after preparation

5. Reflux Alkaline Gastritis (Biliary Gastritis):

- **Definition:**

- Reflux of bile – pancreatic juices → stomach → gastritis.

- **C/P:**

- Bile stained vomiting
- Burning pain not relieved by antacids.

- **Complications:** Cancer stomach.

- **Investigations:**

- Endoscope + biopsy. - Modified HIDA scan.

- **TTT:**

- Bile chelator (Cholestyramine)
- If failed → fashioning of a Roux-en-Y anastomosis.

6. Diarrhea:

- **Causes:**

- Rapid gastric emptying.
- Vagal denervation of small intestine.
- Malabsorption of bile salts.

- **Treatment:**

- Antidiarrheal drugs.
- Cholestyramine.
- Reversed jejunal segment.

7. Gall bladder stones:

– Truncal vagotomy → GB stones → gall stasis.

8. Increase risk of gastric carcinoma after 10 years.

Tumors of The Stomach

Benign	Malignant
• Adenomatous polyp. • Leiomyoma (COMMONEST). • Neurofibroma. • Polyps of Peutz Jegher \$.	• Carcinoma. • Non-Hodgkin Lymphoma. • Leiomyosarcoma.

Benign Tumors

- It can give vague symptoms and appears as a smooth filling defect in barium meal.
- It can bleed or may perforate the stomach.
- May give rise to an epigastric mass
- Malignant transformation can occur

Carcinoma of the Stomach

Incidence

- Males > 45 yrs.
- Second common GIT cancers.

- ▶ **Etiology:** benign lesion, chronic irritation, hereditary
- ▶ **C/P:** ♂ > 40 yrs with dyspepsia, cachexia, mass, obstruction, metastasis
- ▶ **Invest.:** endoscopy & biopsy, Ba meal
- ▶ **TTT:** - **Operable:** total radical gastrectomy
- **Inoperable:** resectable, irresectable

Predisposing factors

Chronic lesion	Chronic irritation	Hereditary
1. <i>H pylori</i> induced. 2. atrophic. 3. Biliary gastritis - Postgastrectomy - Postvagotomy + pyloroplasty 4. Benign gastric tumors 5. chronic GU	- Spicy food. - Spirits. - Smoking. - Smoked fish, ↑salt intake. Smoke → 3,4 benzopyrine. (Food rich in nitrates) → N-Nitrosamine compounds	- Family history - Female > Male - Fatal - Blood group A - Bad prognosis - Associated with atrophic gastritis. - Pernicious anemia.

Pathology

I- Macroscopic			
Site			
Lower 1/3 (Pylorus)	Middle 1/3		Upper 1/3
60%	15%		25% (↑nowadays up to 60%)
Types			
	1- fungating Cauliflower mass	2- Ulcer with raised everted edge	3- Infiltrating (Linitis plastica)
Site	Body & fundus	Pylorus or lesser curvature	Starts in antrum → extends upward → involve the stomach diffusely
Gross	▪ Cauliflower mass with ulceration, necrosis, hemorrhage& infection ▪ Malignant ulcer with raised everted edge & necrotic floor		marked thickening of the wall and reduction of the lumen with intact mucosa
Differentiation	Well differentiated, spreads slowly	Moderately differentiated	Poorly differentiated
4- Colloid type:			
All layers of the stomach are infiltrated by areolar tissue containing transparent gelatinous substance. It is the commonest cause of Krukenberg tumor of ovary.			
II- Microscopic			
Adenocarcinoma 95%		Squamous cell carcinoma 4%	Anaplastic 1%
Columnar cell adenocarcinoma	Colloid adenocarcinoma (Bad prognosis)	Site - Cardia - Fundus Due to metaplasia	Undifferentiated tumor

► Recently pathologists classify gastric cancer into (Japanese classification):

A. Early gastric cancer:

- Limited to mucosa and submucosa.
- May be protruding, superficial or excavating (penetrating).
- Diagnosed only if screening programs are done.
- 5 years survival 90%.

B. Late gastric cancer:

- Involves muscularis mucosa.
- 5 years survival 10%.

This is the commonly diagnosed type in clinical practice

Spread

1. Direct:

- Tumor infiltrates the duodenum, esophagus, surrounding organs.

2. Blood:

- Mainly → liver via portal circulation.
- Rarely → lungs, bones, brain via systemic circulation.

3. Lymphatic:

- The proximal half of the stomach drains to the left gastric and to the splenic LN
- The antrum drains into the right gastric and subpyloric LN
- The greater curvature drains to gastro-epiploic LNs

■ From the previously mentioned groups of LNs, lymphatic drainage end in celiac or superior mesenteric LNs

- Late: to Virchow's LNs (Troisier's sign).
- Spread to porta hepatis is by retrograde lymphatic spread.

4. Transcelomic: either by:

- Seeding.
- Retrograde peritoneal lymphatic spread:
 - *Krukenberg's tumor*: ovarian secondaries occur in younger females when the ovaries are not yet atrophic.
 - *Blumer's shelf* (metastasis to Douglas pouch).

Staging

TNM Classification

➤ Primary tumor:

- T₁** : Confined to mucosa & submucosa.
- T₂** : Extend to serosa without penetrating it.
- T₃** : Penetrates serosa without invasion of adjacent structures.
- T₄** : Invading the surrounding structures.

➤ Regional lymph nodes:

N₁ (perigastric LN):

- Rt. Cardiac LN.
- Lt. cardiac LN.
- Lesser curve.
- Greater curve.
- Suprapyloric.
- Infrapyloric.

N₂ (extraperigastric LN):

- Lt. gastric LN.
- Common hepatic LN.
- Celiac LN.
- Splenic hilar LN.
- Splenic LN.

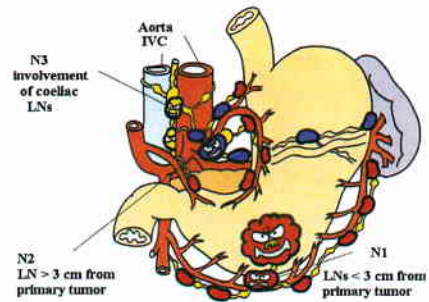
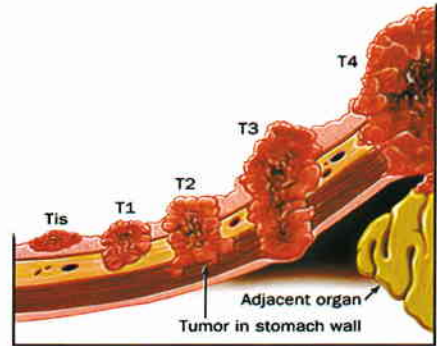
N₃ (LN related to adjacent organs):

- Blood spread: liver.
- Transcelomic spread.

➤ Distal metastasis:

M₀ : No distal metastasis

M₁ : Distal metastasis.



Clinical Picture

► **Male > 40 yrs, with unexplained dyspepsia resistant for TTT for more than 2 weeks**

- The disease is usually diagnosed at a late stage because of the vague and mild symptoms
- Gastric anti-secretory drugs will improve symptoms of gastric carcinoma.
- Patients with carcinoma of the stomach may fall into one or more of 5 groups:

I. Dyspepsia group:

- Usually, male above 40 years of age complaining of dyspepsia, anorexia and has a vague sense of discomfort after meals not responding to medical treatment > 2 weeks.
- Epigastric pain may occur and in late cases may be severe.
- Nausea and early satiety are common.
- A particular dislike to **meat** is often described but is only rarely seen.

2. Cachexia group: (insidious group)

- Loss of weight, asthenia & anemia.
- Unfortunately, a large number of patients would complain from these vague symptoms trying tonics and digestives while their disease progresses to an advanced stage.
- Anemia in the elderly should raise possibility of malignancy and stomach is one of the commonest

3. Mass group:

- An epigastric mass, about **30% of these patients are inoperable.**

4. Obstructive group:

- At the cardia → dysphagia.
- At the pylorus → vomiting.

5. Metastatic group:

- A hard irregular liver due to secondaries, jaundice, malignant ascites
- Enlarged Virchow's LNs (Troisier's sign).

Bleeding → hematemesis and melena
Perforation

Uncommon presentations and usually after TTT
due to necrosis of the tumor

- *Early (curable) gastric carcinoma has no specific features that can differentiate it from benign dyspepsia.*

DD

From other causes of dyspepsia:

1. Peptic ulcer.
2. Biliary dyspepsia of GB diseases.
3. Pancreatic dyspepsia from chronic pancreatitis
4. Colonic dyspepsia from chronic colitis.
5. Appendicular dyspepsia from chronic appendicitis

From other causes of epigastric mass: see later

From other causes of pyloric stenosis: see later

Investigations

- *Liberal use of gastroscopy in patients > 40 years with a new or persistent dyspepsia with biopsy from any suspicious lesion.*

I. For diagnosis:

- Upper GI endoscopy:
 - To visualize the macroscopic picture.
 - To investigate predisposing factors.
 - To take biopsy (punch or brush cytology).
- Barium meal: Shows (accuracy 75%)
 - A- If cauliflower mass:
→ *Irregular filling defect*
 - B- If malignant ulcer:
→ *Ulcer niche*
 - > 2 cms.
 - Outside the ulcer bearing area.



Ulcer niche of a malignant gastric ulcer



Garden's meniscus sign

→ **Linitis plastica** (leather bottle stomach): The stomach appears as a narrow irregular tube without interruption to be examined in Trendelenburg's position.

2. For staging:

1. Abdominal U/S:

- Detects liver secondaries.

2. CT scan:

- Detects LN involvement and helps in staging of the disease.

3. For follow up:

➤ Tumor markers:

- CEA ↑ in 65% and indicates extensive spread.
- It is of prognostic value rather than diagnostic
- CA 19-9.
- CA 72-4 (more recent and more specific)

4. For preoperative preparation:

1. CBC:

- Anemia (micro or macrocytic anemia)

2. KFTs, LFTs, serum electrolytes:

- For operable fitness.



Marked narrowing of almost the complete stomach, due to diffuse infiltration of the gastric wall by a carcinoma (**linitis plastica**)

Treatment

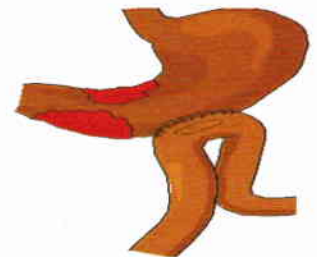
A. Inoperable Cases

➤ Resectable:

Palliative partial gastrectomy (as it is adenocarcinoma → not sensitive to irradiation or chemotherapy).

➤ Irresectable:

1. Pyloric obstruction → **Palliative gastrojejunostomy** or **expandable metal stent**.
2. Cardiac obstruction → **Celestin tube** or esophagojejunostomy



Palliative Gastrojejunostomy

B. Operable Cases

- Good general condition.
- No wide local metastasis.
- No blood or lymphatic spread.

A. English school:

⇒ Upper 1/3:

1. **Esophagogastrectomy**. Through thoraco-abdominal incision
2. **Anastomosis by esophagojejunostomy by Roux-en-Y**.

⇒ Middle 1/3:

1- Total radical gastrectomy → Removal of

1. Stomach with safety margins 5 cm above it and 1.5 cm of duodenum below it
2. Greater & lesser omenta.
3. All draining LNs.
4. Spleen.
5. Tail of the pancreas.

2- Esophagojejunostomy: By Roux-en-Y

⇒ **Lower $\frac{1}{3}$:**

1- **Lower radical partial gastrectomy** (as partial gastrectomy + remove LNs).

2- **Anastomosis** by Polya or Polya with a valve

B. Japanese school:

Total radical gastrectomy whatever the site of the tumor.

Prognosis

- Depends on many factors, the most important are the histological type and the differentiation, the depth of penetration of the wall and the presence or absence of LNs involvement.
- Only about 40% of patients are operable at time of laparotomy.
- 5 yrs survival rate after resection = 5 %.

Gastrointestinal stromal tumors (GIST), formerly leiomyoma

1. Common in the stomach and small intestine. It arises from **Cajal cells** that lie within the stomach wall.
2. May present by abdominal mass, hematemesis or dyspepsia.
3. May reach a large size.
4. May have a pseudocapsule and recurrence is common after surgery.
5. It is treated by local gastric resection.
6. Gleevec is tumor specific monoclonal antibody against some tyrosine kinases of the tumor. It has shown excellent clinical results.

DD of Pyloric Obstruction

Etiology

A- In infants & children:

- 1- CHPS
- 2- Corrosives.
- 3- FB

B- In adults:-

1. Chronic DU.
2. Pyloric canal ulcer.
3. Malignant obstruction.
4. Others as Crohn's disease, TB, sarcoidosis, enlarged LNs & bezoars.

Clinical Picture

Symptoms

- 1- Vomiting (projectile, not bile stained).
- 2- Constipation.
- 3- Abdominal discomfort and sense of fullness.

Signs

➤ General:

- Dehydration, underweight, chest infection.

➤ Local:

- Upper abdominal distension.
- Visible peristalsis from Lt. to Rt.

+ Clinical picture of the cause

Investigations

1. Laboratory:

- **CBC:** anemia.
- **KFTs:** pre-renal failure.
- **Serum electrolytes:** decrease Na^+ , K^+ and Cl^- .
- **Tumor markers:** if suspecting carcinoma.

2. Radiological:

- **Barium meal:**
 - Benign obstruction → dilated stomach & delayed emptying.
 - Gastric cancer → irregular filling defect.
- **Abdominal U/S:**
 - CHPS → thickening of pyloric muscle + dilated stomach.
 - Gastric cancer → liver secondaries.
- **Chest X-Ray:** chest infection.

3. Instrumental:

- **Endoscopy:** stenosed pyloric ring or take biopsy if suspecting malignancy.

Treatment

Preoperative Preparations

- 1- NG suction and washing.
- 2- IV alimentation.
- 3- Correction of fluids, electrolytes (Na^+ , K^+ , Ca^{2+} , and Cl^-) and acid-base balance (alkalosis).
- 4- Chest physiotherapy and antibiotics.

Specific Treatment

According to the cause

- 1- CHPS → Ramsted's pyloromyotomy.
- 2- Healed DU → truncal vagotomy + gastrojejunostomy.
- 3- Gastric cancer → see before.

Acute Gastric Dilatation

Definition

- A condition where the stomach loses its tone (mostly postoperative due to reflex sympathetic overactivity → hypotonicity), so it rapidly dilates to huge size.

- ▶ **Etiology:** surgery, immobilization, aerophagia
- ▶ **C/P:** effortless vomiting, silent abdomen
- ▶ **Comp.:** shock, alkalosis, electrolyte imbalance
- ▶ **Invest.:** for comp. (never Ba meal)
- ▶ **TTT:** - **prophylactic:** Ryle
- **Definitive:** R&M, O_2

Sex

- Males > females.

Predisposing Factors

- Abdominal surgery (rough manipulation of the stomach).
- Child birth.
- Prolonged immobilization.
- Aerophagia (hysterical).
- Opiates & narcotics due to delayed gastric emptying.

Etiology

Unknown

- Gastroduodenal ileus.
- ↑ sympathetic tone.
- Toxins which affect the neuromuscular mechanism.

Pathology

- Stomach is hugely dilated & distended by gases
- Fluids derived from intravascular compartment by back diffusion.
- Intestine is collapsed, empty.
- Dehydration & metabolic alkalosis.

Complications

- Shock.
- Alkalosis.
- Electrolyte imbalance.
- Respiratory complications.

Clinical Picture

Symptoms

2-3 days after surgery

- Hiccups.
- Upper abdominal discomfort
- Effortless vomiting of large amounts of dark watery fluid.

Signs

➤ **General:**

- Dehydration.
- Tachycardia.

➤ **Local:**

- Inspection → Abdominal distention.
- Palpation → succussion splash.
- Auscultation → Silent abdomen.

Investigations

- Blood chemistry.
- NEVER barium meal as it worsen the condition.

Treatment

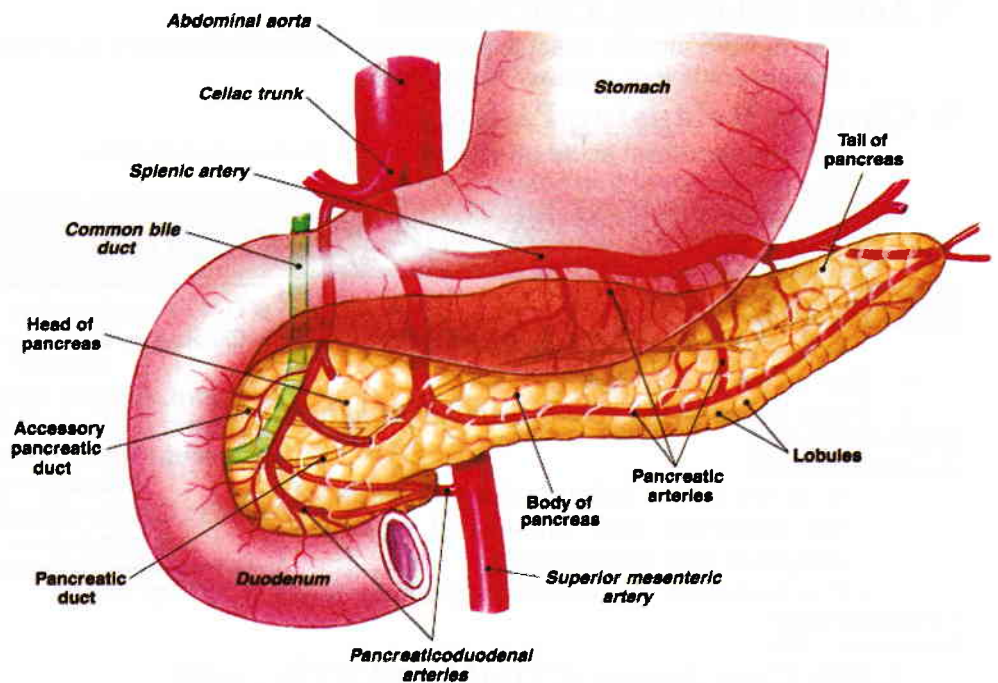
Prophylactic

- Ryle after major abdominal surgeries to prevent overdistension.

Definitive

- Resuscitation + Monitoring.
 - O₂.
 - To be continued until spontaneous cure.
 - No role for surgery.
-

CHAPTER 4



THE PANCREAS

Pancreatitis

Acute Pancreatitis:

- There is severe upper abdominal pain, nausea & vomiting.
- Elevated serum amylase.

Chronic Pancreatitis:

- There is chronic pain.
- Pancreatic calcification on plain X- Ray.
- Exocrine (steatorrhea) & endocrine (diabetes) insufficiency.

Acute Relapsing Pancreatitis:

- It is multiple attacks of acute pancreatitis without permanent scarring.
- The patient is free between the attacks.

Chronic Relapsing Pancreatitis:

- Denotes recurrent acute attacks on top of chronic pancreatitis.
- The patient is not free between the attacks.

A. Acute Pancreatitis

Incidence

- 3 % of all cases of abdominal pain
- Can occur in any age.
- Peak incidence in young men & old women

Definition

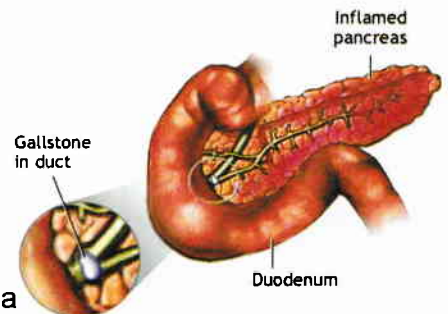
- It is an acute inflammatory disease of the pancreas with release of digestive enzymes and autodigestion.
- It is a serious condition that leads to death in 10% of cases.

- ▶ **Def.:** Autodigestion of the pancreas
- ▶ **Etiology:** ①BD stones(50%) ②Alcohol (35%)
③ERCP ④Idiopathic(20%)
- ▶ **Symp.:(major)** epigastric pain→ back
- ▶ **Signs:** Cullen's, Grey Turner, signs of MOF
- ▶ **Invest.:** Serum (amylase & lipase), CT scan
+ cause + comp.
- ▶ **TTT:** Conservative(6R), surgery,
treatment of the cause

Etiology

1. Bile Duct Stones (COMMONEST[□]): (50%)

- 50% of cases of acute pancreatitis are associated with CBD stones.
- Transient obstruction of the ampulla of Vater which is a common channel between CBD and the pancreatic duct → bile enters the pancreatic duct → activates the pancreatic enzymes.
- It is now realized that, the simple passage of a stone through the ampulla may initiate an attack.



2. Excess Alcohol Intake: (35%)

- Its metabolites activate pancreatic enzymes.

3. Idiopathic Pancreatitis: (20%)

- However, it has been suggested that most of these cases are due to the passage of minute stones through the sphincter of Oddi.

4. Postoperative (Iatrogenic) Pancreatitis:

- CBD exploration & ERCP (1-3%)
- Splenectomy (injury of the tail) → pancreatic fistula.
- Gastrectomy.
- Afferent loop obstruction (after gastro-jejunostomy).



Laparotomy caused acute pancreatitis transformed to pancreatic fistula and clear irritant fluid leaks through draining hole

5. Neoplasm: Peri-ampullary carcinoma.

6. Other rare causes:

- Hypercalcemia (hyperparathyroidism).
- Hyperlipidemia.
- Familial pancreatitis..
- Drug induced pancreatitis:
 - E.g. Corticosteroids, estrogen containing contraceptive pills, Thiazide diuretics, azathioprine & tetracycline.
- Viral → Mumps.

- External injury is not a frequent cause due to the retroperitoneal position of the pancreas, other organ injuries should be excluded. CT & ERCP are done to exclude injury to the pancreatic duct. Surgery is indicated only if there is disruption to the main pancreatic duct.

Pathology

- In acute pancreatitis there is premature activation of the pancreatic digestive enzymes. This leads to auto digestion of the gland .
- The peritoneum is full of a abundant protein rich exudates .
- The enzyme lipase splits fat in the omentum and peritoneum producing glycerol and fatty acids, this acid binds with calcium forming insoluble calcium soap which manifests as white spots of fat necrosis.
- The binding with calcium is likely to produce hypocalcemia in severe cases.
- The proteolytic activity of the liberated enzymes produces vasoactive kinins and other chemical mediators which are responsible for the hemodynamic and respiratory effects (systemic inflammatory response syndrome ; SIRS)



Acute hemorrhagic pancreatitis

Clinical Picture

- Male 50 – 60 years of age with or without history of biliary dyspepsia following heavy meal, alcohol intake or trauma [iatrogenic], the patient complains of major symptoms as local signs are not severe.

Symptoms

- 1- **Severe epigastric pain:** crescendo in character, not responding to usual doses of analgesics, **radiating to the back** [□], relieved in early cases by the **prayer's position** (decreased compression on sympathetic nerves).
- 2- **Repeated vomiting.**
- 3- **Hematemesis & melena:** due to stress ulcer.
- 4- **History of the cause**

Signs

A. General:

1. **Fever & tachycardia** (suppurative phase).
2. **Signs of complications (multiorgan failure) as:**
 - a. **Cyanosis:** due to release of Interleukin-6 & cytokines in large amounts
→ **adult respiratory distress syndrome (ARDS).**
 - b. **Shock:** (Neurogenic, hypovolemic or septic):
→ Caused by hypovolemia & circulating kinins (vasodilators released in septic shock).
 - c. **Jaundice.**

B. Local:

► Local signs are less impressive than expected from the severity of pain. This is due to the position of the pancreas being a retroperitoneal organ lying far from the sensitive parietal peritoneum i.e. it is a disease of maximum symptoms and minimal signs.

1. Inspection and palpation:

- 1- Mild tenderness and rigidity (board like rigidity).
- 2- In late cases:
 - a. **Cullen's sign** →
Bluish discoloration around umbilicus.
 - b. **Grey Turner's sign** →
Bluish discoloration of both flanks.
- 3- Two to three weeks later, an upper abdominal swelling may be observed "pseudopancreatic cyst".



Cullen's Sign

2. **Percussion:** Shifting dullness.

3. **Auscultation:** ↓ Intestinal sounds.

DD

Of acute upper abdominal pain

- 1) **Acute cholecystitis** → epigastric pain radiating to Rt. shoulder & back (Boa's & leak signs).
- 2) **Acutely perforated PU** → Peptic dyspepsia.
- 3) **High small intestinal obstruction** → absolute constipation, ↑ intestinal sounds & on X-Ray → multiple fluid levels.
- 4) **Dissecting aortic aneurysm.**
- 5) **Acute mesenteric vascular occlusion (MVO)**
- 6) **Acute inferior wall myocardial infarction (MI)**

Complications

A. Systemic complications: "MOF" multiorgan failure

1. Shock:

- a- **Hypovolemic** shock (vomiting, exudates & hemorrhage).
- b- **Neurogenic** shock (pain).

2. **ARDS:** Adult Respiratory Distress Syndrome and respiratory failure.

3. **Renal failure:** (hypovolemia or as a part of MOF).

4. **Pleural & pericardial effusion**

5. **DIC:** Consumptive coagulopathy.

6. **Acute gastroduodenal stress ulceration:** & hemorrhage & paralytic ileus.

7. **Tetany:** (due to hypocalcemia).

8. **Lt. sectorial portal hypertension:** splenic vein thrombosis.

B. Local complications:

1. Pseudopancreatic cyst
2. Pancreatic abscess:
 - The patient presents with a swelling in epigastrium & hectic fever.
 - The abscess should be drained.
3. Chronic pancreatitis.

Investigations

1. For diagnosis:

1. Serum Amylase: (*non specific* ☒):
 - a. Elevated within few hours.
 - b. Remains elevated for 2 – 3 days.
 - c. Diagnostic level: > 1000 somogyi units.
 - d. Normal level: 100 – 300 somogyi units.
 - e. Serum amylase is not specific, but in other diseases, it does not exceed 500 units.
 - f. After 5 days, level of serum Amylase drops, but urinary Amylase increases.
 - g. Urinary amylase & amylase/creatinine clearance ratio add little to the diagnostic accuracy.
2. Serum Lipase (specific ☒).
3. CT scan:
 - CT is more accurate ☒ (diagnosis → edema, abscess, cyst, tissue necrosis, & fluid collection in peritoneum).
4. Plain X-Ray of the abdomen:
 - a) Gall bladder stone.
 - b) Colon cut off sign ☒: distended transverse colon and collapse of descending colon.
 - c) Sentinel loop ileus ☒ → distention of the duodenum & upper part of jejunum.
5. Paracentesis :
 - Hemorrhagic fluids & pancreatic enzymes.
6. Laparotomy
7. ECG & cardiac enzymes to exclude MI

2. For the cause:

Abdominal U/S:

- May show gall stones.

3. For complications:

1. CBC:
 - Leucocytosis.
 - Hematocrit value: usually increases due to fluid loss but may decrease in hemorrhagic pancreatitis
2. ABG:
 - To detect cases who might need mechanical ventilation
3. LFTs:
 - ↑ bilirubin due to stone or edema.
4. KFTs:
 - (Amylase / creatinine clearance ratio if > 30% → acute pancreatitis as there is ↑ amylase & ↓ renal function)
 - Renal failure due to hypovolemia or MOF.

5. **Fasting blood sugar:**

- ↑ due to ↓ insulin.

6. **Serum Ca:**

- Decreases due to formation of Ca soaps, ↓ aluminum.

7. **Hypoproteinemia.**➔ **Investigation of acute pancreatitis should be aimed to answering 3 Qs?**

1. Is the diagnosis of acute pancreatitis is correct?
2. How severe is the attack?
3. What is the etiology?

Treatment**Essentially conservative**☑

Resuscitation + Monitoring + No drugs are of value + limited indications for surgery.

A. Conservative (best):

- Severe cases are admitted to the ICU.
- The treatment aims to support different body systems.
- Treatment is simply remembered as the "R" regimen:
 1. **Relief of pain:**
 - By pethidine combined with atropine to avoid spasm of sphincter of Oddi.
 - Morphine should be avoided as it causes severe spasm of sphincter of Oddi.
 2. **Replacement of the lost fluids:**
 - By crystalloids, plasma and blood (in hemorrhagic causes).
 - Monitored by vital signs, urine output and CVP.
 3. **Rest of the pancreas and bowel:**
 - NPO, NG suction.
 - Somatostatin: ↓ GIT secretion.
 4. **Respiratory support.**
 - O₂ mask or mechanical ventilation.
 5. **Resistance of Infection:**
 - By prophylactic antibiotic e.g. imipenem or 3rd generation cephalosporine
 6. **Reassessment after improvement:**
 - By ERCP to remove any residual stones.
 7. **Other therapy:**
 - PAF antagonist e.g. Lexipasant.
 - IV calcium.
 - Diuretic.

B. Surgical:- **Indications of Surgery:**

- 1- If explored after doubtful diagnosis → drain & close the abdomen.
- 2- In late cases; we remove the necrotic tissues which were detected by CT.
- 3- **Complications:**
 - Left-sided portal hypertension → Hassab's operation.
 - Pseudopancreatic cyst → Cystogastrostomy or Cystojejunostomy.
 - Percutaneous aspiration of fluid collection.

C. Treatment of the cause:

- e.g. CBD stones

Bad Prognostic Markers in Acute Pancreatitis (Ranson's criteria)

On Admission			Within the First 48 hrs.		
Age	>	55 years.	Base Deficit	>	4 mEq/L
Fasting blood sugar	>	200 mg%	Estimated fluid sequestration	>	6 L
WBCs count	>	16 000/mm ³	Serum Ca	<	8 mg%.
Serum LDH	>	350 IU/ml ²	HCl decrease	>	10%.
SGOT	>	250 U%.	BUN	>	5 mg%.
			Arterial O₂	<	60 mmHg

- In case of CT guided aspiration and culture of necrotizing pancreatitis revealing E.coli, the most appropriate treatment is exploratory laparotomy and proceeding by excision of the necrotic tissue and 2 tube drains are left.

B. Chronic Pancreatitis

Definition

- It is a medical disorder characterized by chronic inflammation with irreversible impairment of pancreatic function.

Etiology

- Persistent alcoholic consumption.
- Diseases of the common bile duct eg: stone

Clinical picture

- Loss of the exocrine and /or endocrine function of the pancreas
 - Pain** :recurrent attack of epigastric pain
 - DM, Malabsorption**

Investigations

► Radiological:

- X-ray:** shows calcification of the pancreas
- ERCP:** Alternating stricture and dilatation of pancreatic duct {chain of lakes}

Treatment

- Abstinence from alcohol intake is essential

A. Conservative

- Correction of malabsorption by pancreatic extract, H₂ receptor antagonist
- Management of DM by diet and insulin
- TTT of pain by analgesic and stop alcohol

B. Surgical

- Distal pancreatectomy if inflammation is limited to tail
- Whipple operation if the head is to be removed

Pancreatic pseudocyst

Pathology

■ **Nature :**

- This is a collection of pancreatic secretion and inflammatory exudates within a lining of inflammatory tissue rather than epithelium .

■ **Etiology :**

- Develops in 10% of cases of acute pancreatitis , usually after 2-3 weeks .the next common cause is pancreatic trauma

■ **Site :** commonly located in the lesser sac between the pancreas and the stomach

■ **Complications :** infection – Hemorrhage & rupture

- ▶ **Etiology:** acute pancreatitis, trauma
- ▶ **C/P:** asymptomatic, pain, jaundice
- ▶ **Invest.:** Ba meal, U/S, CT
- ▶ **TTT:** spontaneous recovery (drainage if > 6 weeks)

Clinical Picture

A small pseudocyst is painless and may be discovered by follow up U/S.

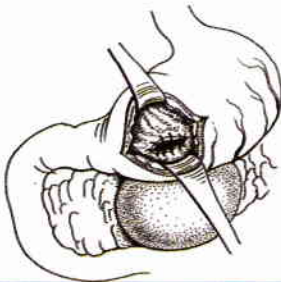
A large cyst causes discomfort and manifests as an upper abdominal swelling

Investigations

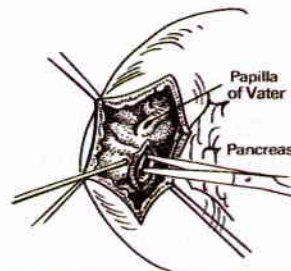
- Barium meal : shows forward displacement of the stomach in a lateral view
- The most accurate diagnostic measures are U/S & CT scan

Treatment

- Most of cases resolve spontaneously
- A persistent cyst that doesn't resolve in 6 weeks is surgically drained. The cyst is internally drained to the stomach or to a jejunal loop



Cystogastrostomy



Cystojejunostomy

Tumors of the Pancreas

A. Exocrine: (*common*)

- **Benign:** Adenoma.
- **Malignant:** Adenocarcinoma & cystadenocarcinoma.

A. Endocrine (*rare*):

- Arise from APUD cells; they are termed apudomas neuron specific enolase (NSE) is the universal tumor marker for these cells.
- There is possibility of associated APUD neoplasms in other organs forming MEN \$.

➤ **Insulinoma:**

a) **Pathology:**

- Tumors of Beta cells of Islets of Langerhan's.
- Usually benign and rarely malignant.

b) **C/P:**

Whipple's Triad[□]:

1. Symptoms of hypoglycemia (sweating, anxiety, tremors, confusion & coma)
2. During the attack, blood glucose level is < 45 mg / dl.
3. Improvement of these symptoms by glucose.

c) **Investigations:**

- 1) ↓ Serum glucose.
- 2) ↑ insulin[□].
- 3) Insulin/glucose ratio > 0.3
- 4) ↑ C - peptide[□].
- 5) Localization of tumor by:
 - CT, selective angiography and selective venous sampling.

d) **TTT:**

1. Enucleation of the tumor
2. Distal pancreatectomy

➤ **Glucagonomas:**

- a) DM.
- b) Nercotizing skin lesions.

➤ **Vipoma (Pancreatic Cholera):**

WDAHA syndrome. (Watery Diarrhea, Asthenia, Hypocalcemia, Achlorohydrria)

➤ **Somatostatinoma:**

- a) DM
- b) Achlorohydrria
- c) Gall stones due to ↓ cholecystokinin.

Investigations

1. **Laboratory:**

- Hormonal assay.

2. **Radiological:**

- U/S, C T & angiography.

Treatment

1. Surgical removal if possible.
2. Symptomatic.

➤ Multicentric tumors:

- Thyroid: Papillary carcinoma.
- Breast: Lobular carcinoma.
- Hepatoma.
- Pancreatic Carcinoma

Zollinger Ellison Syndrome (Gastrinoma)

Definition

- **Type I** → ↑ G cell mass in the stomach.
- **Type II** → Non-beta cell tumor of pancreas secreting gastrin (malignant in 50%).

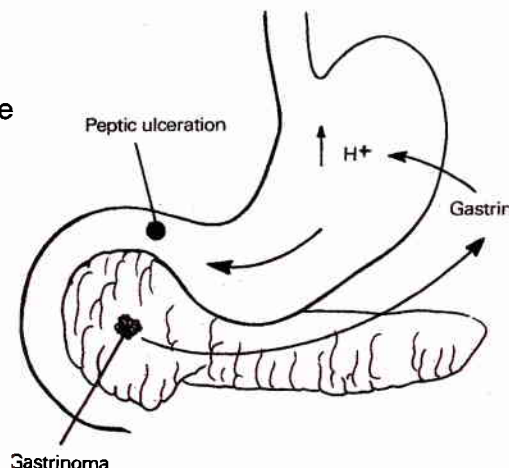
- **C/P:** PU (recurrent, persistent, multiple in extreme of age, diarrhea, steatorrhea)
- **Invest.:** gastrin level, imaging
- **TTT:** detectable or undetectable

Pathology

- 1- Excessive stimulation of parietal cells in the stomach → peptic ulceration.
- 2- Reduction of luminal pH of the Small intestine → inactivation of pancreatic secretion and bile acid → diarrhea & steatorrhea.

Clinical Picture

1. **PU²:**
 1. Resistant for treatment.
 2. Recurrent after treatment.
 3. Multiple.
 4. In ectopic sites (jejunum).
 5. At extremes of age.
2. **Diarrhea & Steatorrhea.** (In 1/3 of cases), due to ↑ intestinal motility by gastrin & inhibition of pancreatic enzymes by the hyperacidity.
3. **Bleeding & Perforation.**



Investigations

1. **Barium meal:** multiple ulcers in unusual sites.
2. **Endoscope:** multiple ulcers in unusual sites
3. **Gastrin level:** by radioimmunoassay:
 - Normal level < 150 ng/L.
 - Most patients show level > 500 ng/L.
 - Gastrin level ↑ by 200 pg/ml > the basal level following secretin injection.
4. **Localization of the tumor** by:
 - CT scan, selective arteriography, selective venous sampling.
5. **Ratio between basal/maximal acid secretion > 0.6**

Treatment

Perform CT and Angiography

✚ If Detectable tumor:

- If in the head → pancreatico-duodenectomy.
- If in the body or tail → distal pancreatectomy.

✚ If Undetectable: (small in size & avascular)

- Blind body & tail pancreatectomy (the tumor is usually present in the body or tail of pancreas)
- Total gastrectomy to remove the target organ.
- Omeprazole in large doses for 6-12months.

Recommended measures for controlling of acid secretion in ZES:

1. Highly selective vagotomy.
2. Total gastrectomy.
3. Vagotomy and pyloroplasty.
4. Medical treatment with Prilosec (omeprazole).

Carcinoma of the Pancreas

Incidence

- Between 55 – 70 years of age.

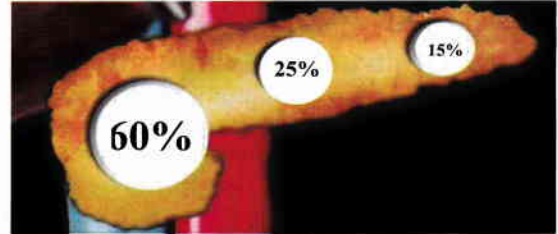
Etiology

- Exact etiology is unknown.
- **Possible causes** : high protein diet – high fat diet – smoking – alcohol

Pathology

- Cancer head 60% : 2/3 of cases in head proper and 1/3 of cases in periampullary region (ampulla of Vater , duodenal papilla & lower end of CBD)
- Cancer body & tail 40%.

- ▶ The commonest affected part is the head
- ▶ Usually arises from acinar & duct cells
- ▶ **C/P**: - old ♂ with rapidly progressive OJ
- Distended palpable GB
- ▶ **Comp.**: obstruction, ascites, LL edema
- ▶ **Invest.**: LFTs, ERCP with biopsy ,
- ▶ **TTT**: a. operable: Whipple's operation
b. Inoperable: triple anastomosis



1. Origin:

- Usually arises from acinar & duct cells.
- From the major duct system as periampullary carcinoma.

2. Macroscopic Picture:

- Mass infiltrating edge, area of hemorrhage and necrosis.

3. Microscopic Picture:

- Usually: Poorly differentiated adenocarcinoma.
- Rarely: Cystadenocarcinoma.

Spread

1. Direct:

- To duodenum, CBD, stomach, transverse colon, peritoneum, aorta, IVC.

2. Lymphatic:

- Celiac LNs.
- Superior mesenteric LNs.

3. Blood Spread:

- Liver (mainly)
- Lungs, bone & brain.

4. Transcelomic:

- Peritoneal nodules, Krukenburg & malignant ascites.

Clinical Picture

- ▶ *Any elderly male with rapidly progressive painless jaundice should be considered as cancer head of pancreas till proved otherwise.*

A. Cancer Head:

(At time of presentation usually larger than 3cm)

1) Obstructive Jaundice☐:

- **Painless** (classic description), but pain is extremely frequent
- **Progressive**:

- Except in periampullary carcinoma, 1 or 2 remissions may occur due to sloughing of a part of the tumor with passage of bile.

- Olive green

2) Distended palpable GB in 50%☐ (Courvoisier's law).

3) Enlarged liver.

4) Asthenia (common).

B. Cancer Body or Tail:

- Much larger at time of presentation (usually irresectable).
- Usually presents late with non-specific symptoms:
 1. **Epigastric pain radiating to the back.**
 2. **Occult Manifestations:**
 - Thrombophlebitis migrans (**Trousseau's sign**☑).
 - Enlarged left supraclavicular LN. (**Troisier's sign**☑).
 - Loss of weight.
 - Metastatic manifestations: jaundice, hepatomegaly, ascites, etc.

DD

Calcular Obstructive Jaundice

Complications

1. **Obstruction leading to:**
 - Malignant obstructive jaundice due to infiltration of CBD
 - Hematemesis or melena, due to:
 - a. Duodenal or pyloric obstruction due to direct infiltration.
 - b. Portal hypertension try to splenic or portal vein thrombosis.
2. **Ascites from liver & peritoneal nodules.**
3. **Edema of the lower limb from IVC obstruction.**

Investigations

1. For diagnosis:

1. LFTs:

- a- **Bilirubin:** ↑serum bilirubin mainly **direct**☑ fraction
- b- **SGOT&SGPT:** no rise unless cholangiohepatitis occurs
- c- **PT:** prolonged due to defect in vitamin K, improved by I.V vitamin K

2. **Stool:** Clay colored ,bulky ,offensive & No stercobilinogen
3. **Urine:** dark colored, frothy ,no urobilinogen& ↑direct bilirubin

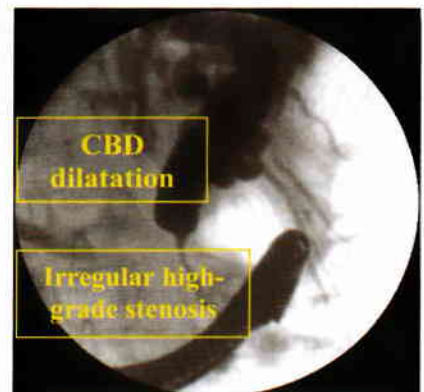
4. **ERCP**(biopsy, stent)

5. **PTC**

6. **Barium meal(obsolete):** widening of the C-curve of the duodenum

7. **Tumor Markers:**

- CA 19-9: recent tumor marker if ↑ indicates irresectable tumor.
- Carcinoembryonic antigen (CEA).
- Pancreatic oncofetal antigen (POFA).
- Pancreatic cancer associated antigen (PCAA).



ERCP
Cancer head pancreas

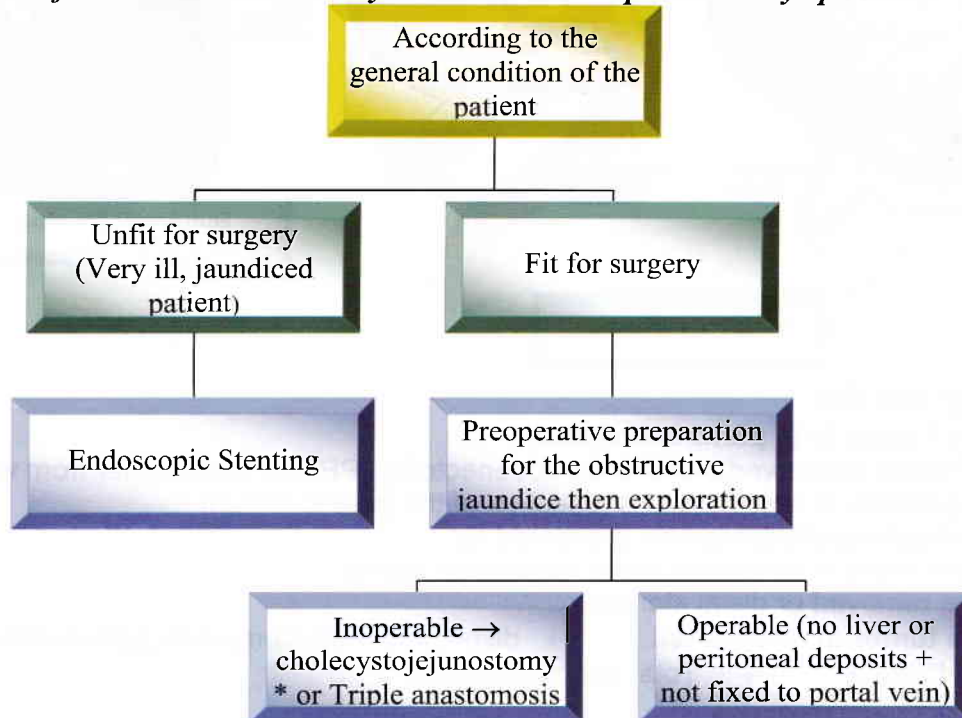
2. For staging:

- **U/S:** endoscopic U/S gives more information about the degree of invasion.
- **CT scan:**
 - Contrast enhanced spiral CT is the most sensitive. It allows targeting the percutaneous FNAC of the lesion.
 - Biopsy is not needed if clinical evidence, elevated CA 19-9 and a hypodense lesion on triphasic CT are all present.
 - When CT appearance is atypical, needle biopsy is considered. Positive cytology justifies resection of operable cases.
 - On the other hand a negative result can not rule out malignancy.

3. For preoperative preparation: • CBC, CXR, KFTs

Treatment

In spite of the limited tumor size the majority of pancreatic head cancers (80%) are not eligible for resection at time of diagnosis. This is due to advanced local tumor extension or the presence of distant metastasis mostly liver metastasis or para-aortic lymph nodes.



* Choledojejunostomy can be done if the gall bladder is unsuitable for anastomosis

Whipple's operation (Pancreatico - duodenostomy)

➤ Removal of:

1. Head & neck of the pancreas.
2. The whole duodenum (as it shares the same blood supply as the head of pancreas).
3. Antrum of the stomach (may not be done in pylorus preserving Whipple's operation).
4. GB & CBD.

N.B.

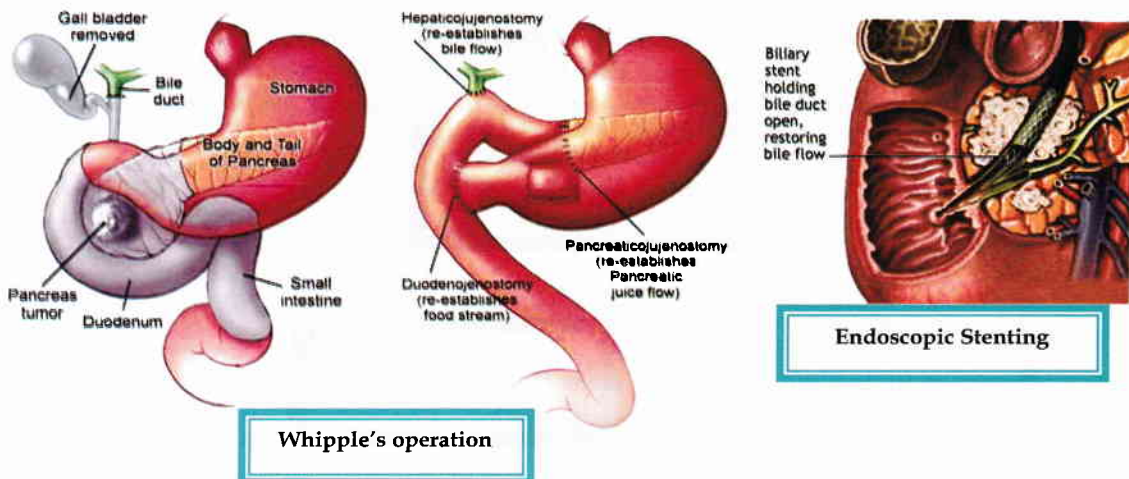
Removal of spleen and body & tail of pancreas (only in total pancreatectomy operation)

➤ The operation ends by the following anastomosis:

1. Hepatic duct to the jejunum.
2. Pancreatic duct to the jejunum.
3. Stomach to the jejunum.

➤ Adjuvant chemotherapy, and radiotherapy:

- May be given after resectional surgery or before operation (neoadjuvant therapy).
- 5-fluoro-uracil and gemcitabine are recommended.



Recently we do

1. If the Tumor is in pancreatic head
 - Pylorus preserved pancreaticoduodenectomy (PPPD) which differ from whipple operation in which there is resection of the gastric antrum
 - whipple operation is now preserved for situations in which the entire duodenum has to be removed or distal stomach
2. If the tumor in body & tail → distal pancreatectomy with splenectomy
3. If multifocal → Total pancreatectomy

Palliative treatment

1. Relieve jaundice + treat biliary sepsis
 - Surgical biliary bypass
 - Stent placed at ERCP or percutaneous transhepatic cholangiography
2. Improve gastric emptying :
 - Surgical gastroenterostomy
 - Duodenal stent
3. Pain relief.
4. Symptom relief for quality of life
 - Enzyme replacement for steatorrhea
 - Treatment of DM
5. Consider chemotherapy

Prognosis

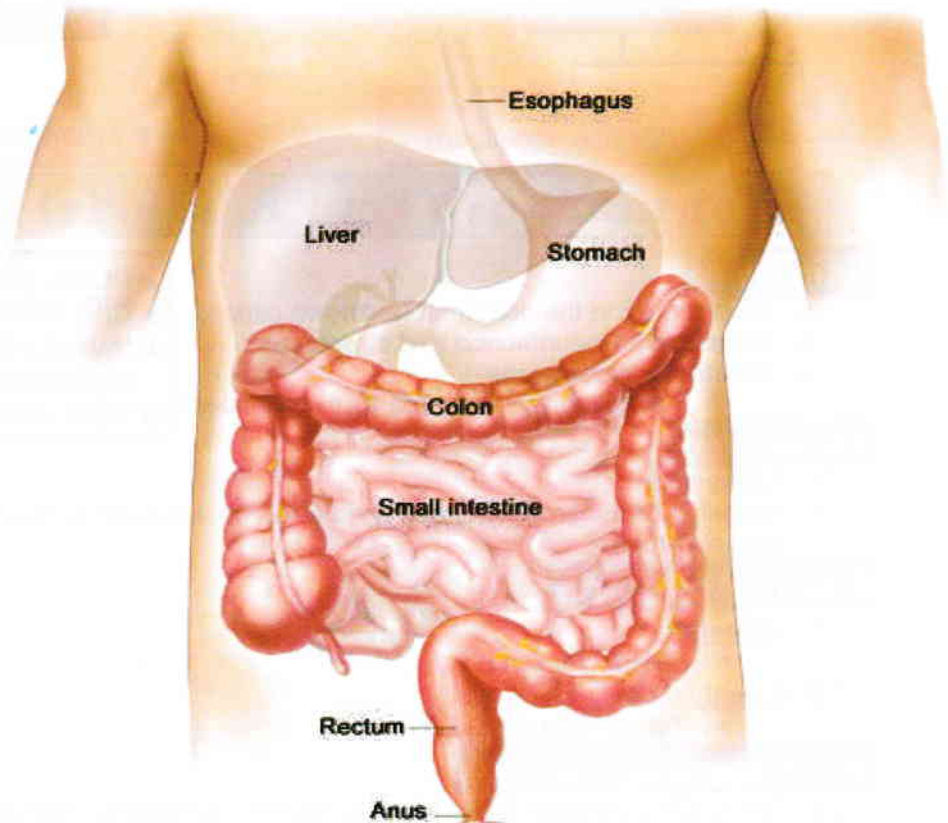
- Extremely poor → 5 years survival rate is < 5 %.

➤ Abdominal pain with ↑ serum amylase:

1. Afferent loop obstruction.
2. Perforated duodenal ulcer.
3. Duodenal stump blowout.
4. Biliary peritonitis.
5. Rupture aortic aneurysm.
6. Disturbed ectopic pregnancy.
7. Mesenteric infarction.
8. Retroperitoneal hematoma.
9. Torsion of an intra-abdominal viscus

} Duodenal causes

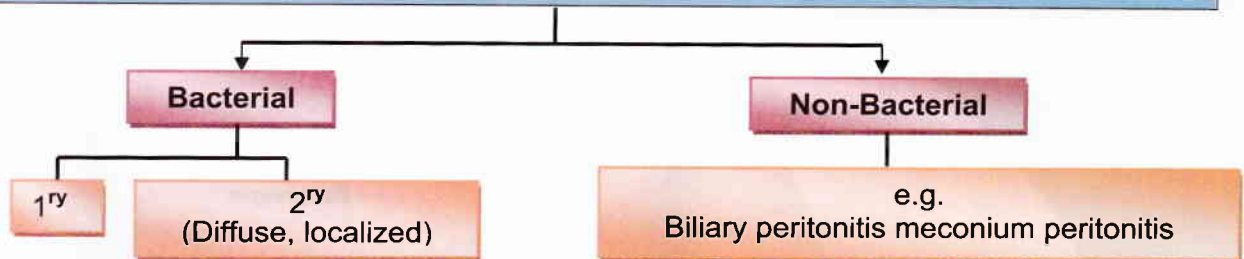
CHAPTER 5



THE PERITONEUM

The peritoneum

Acute Peritonitis



Spontaneous (1^{ry}) Bacterial Peritonitis

Etiology

- No 1^{ry} focus in the abdomen (unknown cause).
- Organism:** Pneumococci and streptococci
- Route:** → Blood borne.
→ Fallopian tube (organism: gonococci).

- ▶ **PDFs:** ↓immunity, ascites, dialysis
- ▶ **C/P:** FAHM, tenderness, rigidity
- ▶ **Invest.:** diagnostic (laparoscopy, aspiration)
- ▶ **TTT:** conservative, drainage

Incidence

- Uncommon.
- Usually affects female children where infection is thought to reach peritoneal cavity through fallopian tubes

Clinical Picture

- General:**
 - FAHM, vomiting.
- Local:**
 - Tenderness & rigidity (mild).

Predisposing Factors

- Immunosuppression (splenectomy, nephritic syndrome, malnutrition, abdominal malignancy).
- Liver cirrhosis with ascites.
- Peritoneal dialysis for chronic renal failure.

Investigations

- Diagnostic laparoscopy.
- Diagnostic aspiration (staining – culture & sensitivity)

Treatment

- **Conservative:** If sure of diagnosis. 3rd generation cephalosporin.
- **Laparotomy & Drainage:** If unsure.

Acute (2^{ry}) Bacterial Peritonitis

Etiology

1- Causative Organism:

- Gram -ve: E.coli (**COMMONEST**).
- Gram +ve: Staph, Strep, Pneumococci.
- Anaerobe: Bacteroids, Clostridium welchii.

2- Route of Infection:

- 1- Direct Spread From :
 - a- Infected organs: e.g. appendicitis, cholecystitis or diverticulitis
 - b- Leaking organs: e.g. perforated PU, leaking anastomosis
 - c- Through an operation or a traumatic wound.
- 2- Blood Spread: may occur in cases of septicemia and pyemia (rare)

Pathology & Fate

1. Resolution if the source of infection is controlled or removed.
2. Localization & abscess formation either around the 1ry viscus or in an anatomical compartment.
 - Factors which facilitate Localization of Infection:
 1. Anatomical:
 - The peritoneal cavity is divided into 2 compartments:
 - 1- Greater sac.
 - 2- Lesser sac.
 2. Peristalsis:
 - Reduced & this helps in preventing spread of the infective process.
 3. Greater Omentum:
 - Becomes adherent to the inflamed structures (policeman of the abdomen).
 4. Peritoneal Fluid:
 - Rich in PNL and antibodies.
3. Flaring up → diffuse peritonitis.
 - Factors which facilitate Diffuse Peritonitis:
 1. Rapid Rate of Infection:
 - e.g. perforated viscus → no time for localization.
 2. Centrally Situated Organs
 - e.g. Mickle's diverticulum (with failure of localization).
 3. Defect in Omentum:
 - Pregnancy → elevated omentum.
 - Children → small omentum.
 4. Enema & Purgatives → ↑↑ peristalsis → spread of infection.
 5. High Virulence of Organism.
 6. Low Patient Resistance.
4. septicemia → death

Diffuse (2ry) Bacterial Peritonitis

Clinical Picture

- ▶ The patient presents in the beginning with the clinical picture of the original cause then presents by:

- ▶ **Etiology:** E.coli, (direct, blood)
- ▶ **C/P:** of the cause then as I.O.
- ▶ **Comp.:** MOF, Ileus, Incisional h., adhesive I.O., Infection, Imbalance
- ▶ **Invest.:** U/S, aspiration, x-ray, KFTs
- ▶ **TTT:** R&M + exploratory laparotomy

Symptoms

- 1- Pain:
 - Persistent, dull aching, increases by movement or coughing
 - The site of maximum pain is at the site of the original lesion.
- 2- Abdominal Distension.
- 3- Vomiting:
 - At first gastric contents are vomited then it becomes bilious.
 - In long standing cases, the vomitus becomes feculent.
- 4- Absolute Constipation:
 - If there is pelvic collection, patient may have tenesmus or diarrhea

Signs

1- General:

- The patient looks distressed, toxic, lies still in bed and avoids any movement.
- There is fever & tachycardia.

2- Local:

1. Inspection:
 - Limited abdominal movements with respiration all over the abdomen
 - Abdominal distension.
2. Superficial palpation: Guarding all over the abdomen and tenderness.
3. Deep palpation: Rebound tenderness all over the abdomen.
4. Percussion:
 - Shifting dullness.
 - Tympanitic resonance (if intestinal obstruction)
5. Auscultation:
 - Absent or diminished intestinal sounds (dead silent abdomen).
6. PR / PV:
 - Tenderness & fullness in Douglas pouch in females
 - Fullness in the rectovesical pouch in males.

► In long standing cases → (facies hippocratica) pale and toxic withdrawn face, sunken eyes, dry tongue and lips, anxious look

Causes of death & complications of Generalized Peritonitis

- 1) Toxemia, septicemia, pyemia, bacteremia.
- 2) Multiple organ failure.
- 3) Paralytic ileus.
- 4) H₂O and electrolyte imbalance.
- 5) Wound infection.
- 6) Incisional hernia and adhesive intestinal obstruction.
- 7) Residual abscess.

Investigations

1. For diagnosis:

- U/S:
 - Intra-abdominal fluid collection.
- Peritoneal diagnostic aspiration:
 - May be helpful in determining the nature of the fluid e.g.:
 - Bilious → perforated DU.
 - Pus → bacterial peritonitis.
 - Serosanguinous → pancreatitis
- CBC:
 - Reveals leucocytosis.

2. For the cause:

- Plain X- Ray abdomen:
 - May indicate the cause (e.g. air under diaphragm in perforated viscus).
 - May demonstrate paralytic ileus with the distension of small and large bowel.
 - Multiple fluid levels in intestinal obstruction.

3. For complications:

- KFTs: Pre-renal uremia.

Treatment

► Preoperative preparation then urgent operation

1. Resuscitation & Monitoring:

A- Resuscitation:

1. Ryle

2. IV line:

- a- Fluids.
- b- Blood transfusion.
- c- Analgesics → for pain (after sure diagnosis).
- d- Combined antibiotics:
 - To cover aerobic and anaerobic organisms.
 - Ampicillin, Metronidazole and Aminoglycoside.

3. Catheter

B- Monitoring: BP, pulse, urine output, CVP, electrolytes and pH.

2. Exploratory Laparotomy:

1- Incision:

- Midline incision in doubtful cases or paramedian

2- Peritoneal toilet:

- (Saline + betadine or antibiotic) then the pus & foreign materials are removed.

3- Identification of the cause:

- It should be dealt with e.g. acute appendicitis → Appendicectomy.

4- Drainage

- Hepatorenal drain: most dependent area while lying down.
- Pelvic drain: most dependent area during standing.
- Drain beside the cause.
- Subcutaneous drain

5- The abdomen is closed.

► Postoperative care better in an ICU to support different systems.

Localized 2ry Bacterial Peritonitis

1- Subphrenic abscess.

2- Iliac abscess.

3- Pelvic abscess.

1. Subphrenic Abscess

It is accumulation of pus under diaphragm.

Anatomical Considerations

- The subphrenic region is considered as the portion of the abdominal cavity which extends from the diaphragm above to the transverse colon and mesocolon below.
- The region is divided by the liver into suprahepatic and infrahepatic compartments.
- The falciform ligament divides suprahepatic compartment into Rt. and Lt. portions.
- The subphrenic spaces, therefore, include the following:

► **Anatomy:** intra & extraperitoneal spaces
 ► **Etiology:** E.coli, inflamed viscus, postop.
 ► **C/P:** FAHM, hiccup, 4 perc. zones
 ► **Invest.:** abdominal (U/S, CT), CXR
 ► **TTT:** AAA, drainage (percutaneous, open)

A- Intra-peritoneal Spaces:-**1- Right Anterior Subphrenic Space:**

- This is the space that lies between:
 - ▶ Diaphragm
 - ▶ Superior and anterior surfaces of the right lobe of the liver
 - ▶ To the **right** of the falciform ligament.

2- Left Anterior Subphrenic Space:

- Bounded by
 - ▶ Diaphragm
 - ▶ Left lobe of the liver, stomach and spleen
 - ▶ To the **left** of the falciform ligament.

3- Right Posterior Subphrenic space: (Hepatorenal pouch of Morrison[□]).

- ▶ Above and in front: liver and gall bladder
- ▶ Below and behind: upper pole of the right kidney, lower part of the right suprarenal gland and the second portion of the duodenum.

4- Left Posterior Subphrenic Space:

- This is the lesser sac.

▶ The commonest space to be affected is the Morrison's pouch.

B- Extra-peritoneal Spaces:-**1- Right Extraperitoneal Space:**

- Lies between the bare area of the liver and the diaphragm.

2, 3 Two perinephric spaces.**Etiology****➤ Causative Organism:**

- E. coli, Staph. or Strept.

➤ Route of Infection:

1. Residual pus collection following generalized peritonitis.
2. Inflamed or perforated viscera e.g. appendix, GB.
3. Postoperative collection after appendectomy or cholecystectomy.
4. Lymphatic spread from chest infection.

Clinical Picture**Symptoms****➤ General:**

- FAHM.

➤ Local:

- 1- Persistent **hiccough**[□].
- 2- **Pain** is slight or absent
 - Site: in the epigastrium or in the Rt. Hypochondrium.
 - Referred to: the shoulder.
 - Increases by: inspiration.

▶ **Subphrenic** abscess should be suspected whenever hectic fever develops or persists after the treatment of any inflammatory lesion within the abdomen

▶ **Pus somewhere , Pus nowhere else, Pus under the diaphragm**

Signs**➤ General:**

- Hectic fever, tachycardia, rapid deterioration of the general condition of the patient, may be pleural effusion.

➤ Local:**1. Inspection:**

- Impaired movement.
- Rarely, there is bulging of the lower ribs or upper abdomen.

2. Palpation:

- Tenderness may be present in the lower ribs and just below the costal margin.
- There may be swelling and rigidity of the upper abdomen.
- Downward displacement of liver with upward displacement of the apex of the heart.

3. Percussion:

- If the abscess contains gas, **4 percussion zones** may be elicited:
 - a. Resonance of the lung.
 - b. Dullness of the pleural effusion.
 - c. Resonance of the gas in the abscess.
 - d. Dullness of the liver or pus in the abscess.

4. Auscultation:

- Decrease air entry and crepitations over the lung base of the affected side.

DD

1. Pneumonia.

2. Liver abscess.

3. Pyelonephritis.

Investigations

1. Imaging:

• Abdominal U/S & CT:

- The best localizing method.

• Chest X- Ray:

- Thickened elevated and fixed copula of the diaphragm (**Tented Diaphragm**)
→ diminished movement of the copula by screen.
- Homogenous opacity obliterating the corresponding costophrenic angle rising to the axilla (**pleural effusion**).
- Localized **gas under the diaphragm** due to :
 - Perforated viscus.
 - Infection by a gas forming organism.

2. Laboratory:

• CBC:

- ↑ TLC & shift to the left.

Treatment

1. Conservative: (early)

- Rest, Analgesics, Antibiotics, Antipyretics.

2. Percutaneous Drainage:

- In case of:
 1. Failure of conservative treatment.
 2. Evidence of suppuration.
- Percutaneous U/S or CT guided aspiration.

3. Open Drainage:

• Indication:

- Abscess with multiple loculi or thick pus or with rapid reaccumulation.

• Techniques:

1- Posterior Abscess:

- **Extrapleural extraperitoneal approach.**
- Subperiosteal excision of 12th rib.
- Transverse incision is done at its bed.
- A finger is inserted to burst the abscess and break all loculi.
- A drain is inserted.

2- Anterior Abscess:

- Subcostal Incision.
- Extraperitoneal approach.
- All layers are divided except peritoneum.
- A finger is inserted to burst the abscess and break all loculi.
- A drain is inserted.

2. Pelvic Abscess

Pathology

- Pus collects in the Douglas pouch or rectovesical pouch.

Fate

- A. Spread → generalized peritonitis.
- B. Rupture in rectum or vagina → spontaneous cure.

Causes

- A. Acute pelvic appendicitis.
- B. Pelvic inflammatory disease (PID) in females.
- C. Localization of diffuse peritonitis.

Clinical Picture

Symptoms

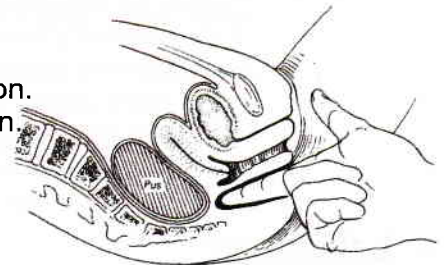
1. **General:**
 - FAHM.
2. **Local:**
 - Dysuria and frequency due to bladder irritation.
 - Tenesmus and diarrhea due to rectal irritation.
 - Pain (pelvic, perineal & suprapubic).

Signs (PR) or (PV)

- Tense, cystic and tender mass (tender boggy).
- If large → felt in the suprapubic region.

Treatment

- **Drainage & put a drain (according to the pointing part):**
 - Colpotomy (vagina).
 - Proctostomy (rectum).
 - Suprapubic (extraperitoneal) midline.



Rectal examination for pelvic abscess

In general, provided the abscess is shut off from the general peritoneal cavity, rectal drainage of a pelvic abscess is preferable than suprapubic drainage, which breaks down nature's barriers and exposes the general peritoneal cavity to the dangers of spreading infection.

3. Iliac Abscess

Definition

- Collection of pus in the iliac fossa.

Etiology

1. **Rt. Iliac fossa** from acute appendicitis and perforated DU.
2. **Lt. iliac fossa** from diverticulitis or ulcerative colitis.
3. **On both sides:** tubo-ovarian abscess, localization of diffuse peritonitis.

Clinical Picture

Symptoms

1. **General:** FAHM.
2. **Local:**
 - Pain in the iliac fossa.
 - Vomiting and constipation.

Signs

- Swelling in the iliac fossa.
- Tenderness in the iliac fossa.

Investigations

- **CBC:** ↑ TLC.
- **U/S.**

Treatment

- Extraperitoneal drainage (muscle cutting incision).
- Nowadays: Percutaneous U/S or CT guided aspiration.

Acute Non-Bacterial Peritonitis

A- Biliary Peritonitis

- **e.g.**
 1. After cholecystectomy (cholecystohepatic ducts).
 2. Leakage around a T-tube.

B- Meconium Peritonitis

⇒ Gut perforation (neonatal – late intrauterine) → aseptic peritonitis.

- **C/P:** Neonatal vomiting + abdominal distension + failure to pass meconium.
- **Treatment:** Laparotomy & suture the perforation.

TB Peritonitis

Etiology

- It is always secondary to a tuberculous focus elsewhere.

- ▶ **Etiology:** 2ry TB, (direct, lymph, blood spread)
- ▶ **Path.:** ascitic, encysted, adhesive, caseous, miliary
- ▶ **Symp.:** TB toxemia, vague abdominal pain
- ▶ **Signs:** doughy abd., rolled omentum (sausage)
- ▶ **Invest.:** as TB + laparoscopy & CBC
- ▶ **TTT:** mainly medical

Route

1. **Direct:** (intestinal TB - tabes mesenterica – TB salpingitis) COMMONEST.
2. **Hematogenous:** e.g. pulmonary TB.
3. **Lymphatic:** e.g. from pleura or bowel.

Pathology

1. **Ascitic:** *commonest*
 - Tubercles + fluid (+) lymphocytes & tubercle bacilli.
 - The greater omentum is thickened and frequently rolled-up forming a **sausage shaped mass** above the umbilicus.
2. **Encysted:** *localized form of ascitic type*
 - The fluid is encysted by fibrous adhesions. It produces an intra abdominal cyst which may be mistaken for ovarian or mesenteric cyst.

3. Adhesive:

- Adhesive Intestinal obstruction.
- Omentum is rolled-up (Mass).

4. Purulent (Caseous):

- Rare → in young females (as a complication of TB salpingitis), cold abscess formation → sinus & fistula.

5. Acute Miliary type:

- It resembles acute peritonitis and the diagnosis is frequently made during operation.
- The exudate is straw colored.
- The peritoneum is studded with tubercles

- Suspect tuberculous peritonitis in a young emaciated patient with a long history of fever, abdominal pain, distension and abdominal tenderness.
- Suspect a pelvic abscess if there general manifestations of pus collection + mucous diarrhoea and burning micturition.
- Suspect a subphrenic abscess in:
 - Persistent pyrexia following an abdominal operation or an inflammatory lesion in the abdomen.
 - General manifestations of pus collection without local symptoms nor signs anywhere

Clinical Picture

► More common in children and young adults

➤ **Symptoms of TB toxemia:**

- Night fever, night sweat, loss of weight & loss of appetite.

➤ **Recurrent attacks of non-specific abdominal symptoms:**

- Pain (vague), colics, diarrhea, distension due to toxemia, TB enteritis.

➤ **Abdominal examination shows:**

▪ **Palpation:**

- Doughy abdomen, generalized tenderness with mild rigidity, palpable mass: rolled omentum "sausage-shaped", TB LNs.

▪ **Percussion:**

- Free ascites (usually small volume).

➤ **PV:** may show tubo-ovarian mass.

Investigations

► The most important diagnostic tools are:

- CBC.
- Laparoscope.

1. Laboratory:

1. **CBC:**

- Anemia, lymphocytosis, high ESR.

2. **Tuberculin test:** +ve.

2. Radiological:

1. **Plain CXR:** may reveal a primary focus.

2. **Abdominal U/S:** ascites free or encysted, LNs, no liver or spleen affected.

3. Instrumental:

1. **Abdominal Tapping:** *Ascitic fluid*

- Straw colored.
- Special specific gravity (> 1020).
- Rich in lymphocytes & proteins.

2. **Laparoscopy:**

- It allows visualization of peritoneum, biopsy from tubercles.
- It is the most reliable method for diagnosis of TB peritonitis

3. **Exploratory laparotomy.**

Treatment *Mainly medical*

- At least 2 antituberculous drugs for at least one year.
- Surgery is indicated in few cases such as (Intestinal obstruction – doubtful diagnosis – cold abscess).

Tabes Mesenterica

(Tuberculous mesenteric Adenitis)

Definition

- It is the childhood type of intestinal TB, which is similar to the primary complex of the lungs (but less common)

- ▶ **Etiology:** contaminated milk
- ▶ **C/P:** TB toxemia, abdominal pain (as appendicitis in acute cases)
- ▶ **Invest.:** CBC, X-ray
- ▶ **TTT:** medical, surgical

Etiology

- A. Organism:** Mycobacterium tuberculosis (bovine type)
- B. Route of entry:** Ingestion of contaminated milk

Pathology

- A small focus in distal ileum (Payers Patches) similar to Ghon's focus in the mid zone of the lung
- Huge enlargement of mesenteric nodes similar to the enlarged mediastinal nodes.

Fate

➔ **Resolution or activation takes place depending on the body resistance**

A. Resolution:

- Healing by fibrosis and calcification
- Later adhesions and kinking may produce intestinal obstruction.

B. Activation:

- Caseation produces a mesenteric cold abscess
- Rupture in the peritoneal cavity produces tuberculous peritonitis
- Spread to thoracic duct may cause military tuberculosis.

Clinical picture**Symptoms**

- 1- **TB toxemia:** low grade fever, pallor, wasting, sweating
- 2- **Abdominal pain** with vomiting and diarrhea.

Signs

- 1- Tenderness below and to the right of the umbilicus
- 2- Firm irregular masses may be felt
- 3- **IN acute cases** there are severe tenderness, rebound tenderness and rigidity in the RT iliac fossa simulating appendicitis

Investigation**1. Laboratory:**

- **CBC:** TLC is normal

2. Radiological:

- **X-ray:** of the abdomen
 - May show calcified nodes as mottled shadows arranged along the line of the small intestinal mesentery.

Treatment

A. Medical:

- Antituberculous treatment is given for active cases

B. Surgical:

- For complicated cases as peritonitis or intestinal obstruction

Differential diagnosis of ascites

	Cirrhotic	Malignant	Tuberculous
Age	Any	Early but may be young in ovarian cancer	Usually young
History	Jaundice or hematemesis	Short history Symptoms of the 1ry	Toxemia
General exam	Liver insufficiency	Distant metastases	Toxemia
Abdominal exam	HSM may be present	Multiple hard abdominal masses may be palpable	Doughy mass may be palpable
LFTs	Poor	Normal	Normal
U/S	Liver cirrhosis/ splenomegaly	Abdominal masses may be detected	Abdominal masses may be detected
Tapping	Transudate	Exudate and cytology may show malignant cells	Straw colored exudates and culture may reveal T.B bacilli
Treatment	- Medical - Peritoneo-venous shunt	- Tapping - Radioactive gold	Anti-tuberculous
Prognosis	Bad	Very bad	good

Mesenteric Cysts

Definition

Fluid collection between 2 layers of the small bowel mesentery

A. True cysts:

[a] Chylolymphatic cyst: *Commonest*

- It is a retention cyst due to obstructed lymphatic drainage.
- It is thin walled, lined by endothelium and contains lymph.
- It has a separate blood supply from that of the related intestinal loop.

[b] Enterogenous cyst:

- From → Sequestered intestinal epithelium.
→ Intestinal duplication (inframesenteric).
- It is thick walled, lined with mucosa & contains mucus.
- Having the same blood supply of the related intestinal loop.

[c] Teratomatous dermoid cyst.

[d] Hydatid cyst.

[e] Cyst of urogenital remnants.

} TTT: enucleation

Symptoms

- [1] Painless abdominal swelling.
- [2] Attacks of abdominal pain (stretch & obstruction of bowel).
- [3] Acute abdomen (complicated cyst) e.g. torsion, rupture, hemorrhage & infection.

Signs

Tillaux Triad

- [1] Cystic swelling near the umbilicus.
- [2] Crossed by band of resonance (bowel).
- [3] Mobile across and not along the root of mesentery.

B. False Cysts:

Without epithelial lining

⇒ e.g.

- [1] Traumatic blood cyst → TTT: evacuation.
- [2] Encysted TB peritonitis → TTT: medical.
- [3] Breaking down LN (TB & lymphoma).

Investigations

1. U/S – CT scan.
2. IVP: (exclude renal cyst).
3. Barium meal: displacement of bowel + narrow segment.
4. Plain X-Ray: calcified hydatid or mesenteric LNs.

Treatment (according to the cause)

- **Enterogenous:**
 - Excision + resection anastomosis of the related loop of intestine.
- **Chylolymphatic:**
 - Cystectomy only.

► *Abdominal cysts that may attain a huge size:*

1. Pancreatic pseudocyst (upper abdomen)
2. Mesenteric cyst (midabdomen, mobile)
3. Ovarian cyst (pelviabdominal)
4. Hydronephrosis (extends to renal angle)

Mesenteric Lymphadenitis

	Acute Appendicitis	Non-specific Mesenteric Lymphadenitis
Course of pain	Continuous	Periods of complete freedom of the pain
Point of maximum tenderness	McBurney's point	Above and medial to the McBurney's point
Rovsing's sign	Positive	Negative
Shifting tenderness	Absent	Present

Treatment

- Usually these patients are operated upon with a provisional diagnosis of acute appendicitis.
- The mesentery is found to be congested and contains multiple soft LNs.
- Appendectomy is performed and the postoperative course is usually uneventful.

CHAPETR 6



THE APPENDIX

Acute Appendicitis

Prevalence

a. It is the **COMMONEST** cause of acute abdomen in young adults and appendectomy is the most frequently performed urgent abdominal operation.

b. **Age:**

- It usually occurs at 20 - 30 years of age.
- ♂ : ♀ = 3 : 2
- Rare in old age due to atrophy of the lymphoid tissue & fibrosis of the appendix with complete obliteration of the lumen.
- If occurs in an old patient, **you must suspect cancer caecum**.
- Rare in children > 5 years of age due to short wide lumen of the appendix → obstruction & stasis does not occur.

c. **Racial Differences:**

- Appendicitis is common in western countries and urban areas in comparison to rural areas due to low fiber diet → constipation → stasis and infection.

d. **Causative Organism:**

- E.coli (85%), staph, Strept. Fecalis.

e. **Route of Infection:**

- Usually direct from the lumen.

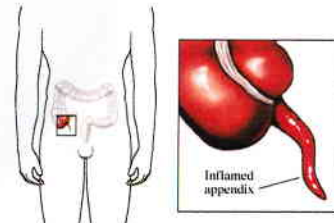
f. **Predisposing Factors:**

a) **Obstruction:** ($\frac{2}{3}$ of the cases)

- It is the commonest & most important predisposing factor.
- It may be due to causes in the:
 - **Lumen:** hard feces, oxyuriasis, ascaris, FB.
 - **Wall:**
 - Rarely tumors of the appendix or caecum.
 - Swelling of lymphoid tissue in response to viral infection.
 - **Outside:** cancer caecum (*in old age*) & adhesions.
- Distension of the obstructed appendix promotes overgrowth of the resident bacterial flora and subsequent invasion of the mucosa.

b) **Anatomical factor:**

- The appendix has a narrow Lumen.



- ▶ **Etiology:** 20-30y., E.coli, direct spread, obstruction, narrow appendix, ♂:♀=2:3
- ▶ **Path.:** obstructive($\frac{2}{3}$), non-obstructive($\frac{1}{3}$)
- ▶ **Comp.:** local, general
- ▶ **C/P:** Typical (shifting abdominal pain, n.&v.) special signs, atypical forms
- ▶ **Invest.:** clinical diag. (TLC, U/S)
- ▶ **TTT:** Appendectomy +TTT of complication

Pathology

⇒ **2 types:**

A. Acute Obstructive Appendicitis ($\frac{2}{3}$ of the cases):

1. **Catarrhal** → mucocoele of the appendix.
2. **Suppurative** → pyocoele or empyema of the appendix
3. **Gangrenous** → (common) occurs at the tip or at the site of obstruction → generalized peritonitis (rapid perforation with no time for omental localization)

B. Acute Non-obstructive Appendicitis ($\frac{1}{3}$ of the cases):

(Common in constipated persons)

1. **Catarrhal** inflammation.
2. **Suppurative** inflammation.
3. **Gangrenous** inflammation (less common) occurs only at the tip → localized peritonitis (delayed perforation allowing omental localization).

Fate

A. The acutely inflamed appendix may resolve, but it is liable for recurrence.

B. Complications :

I. Local complications:

1. Persistent Obstruction → Gangrene and perforation:

- Site:
 - At the tip where the appendicular vessels are very close to the wall and compressed by the edematous appendix.
 - At the site of obstruction due to pressure necrosis.
- Incidence:
 - In children < 36 %.
 - In elderly 44 %.
- Sequelae:
 - Generalized or localized peritonitis depending on:
 - Times between the onset of inflammation and the perforation.
 - Severity of the inflammation.
 - Body defense mechanisms.

2. Appendicular mass (Phlegmon[®]):

- When the inflammatory process is slow (> 48 hours), the body defense mechanism has enough time to surround the inflamed appendix by adhesions to the nearby intestine and omentum. Thus, forming appendicular mass within **3-5 days** after starting of pain.
- It is suspected by history of pain 3 days ago and temperature 39° (EUA is needed to confirm diagnosis).

3. Appendicular Abscess:

- Perforation of the appendix within an appendicular mass forms an abscess.

4. Chronic Appendicitis (Recurrent Subacute appendicitis):

- Usually on top of acute non-obstructive catarrhal appendicitis
- Pathology:
 - Fibrosis of the wall
 - Narrow lumen with kinking and adhesions.
- Clinical Picture:
 - Recurrent attacks of abdominal pain and dyspepsia.
 - Nausea & vomiting.
 - Tenderness in the right iliac fossa.
 - Might be part of Wilkie's triad.
- DD:
 - Irritable Bowel syndrome (IBS).
 - Amoebic colitis.
 - Chronic calculous cholecystitis.
 - Crohn's disease.
- Investigations:
 - Barium enema:
 - Non filling of the appendix.
 - Irregular filling of the appendix.
 - Delayed emptying.
 - Tenderness on screening.

- **III:**

- **Appendectomy:**

- Rt. lower paramedian incision due to adhesions & to explore the abdomen to exclude other causes of dyspepsia.

5. **Fecal Fistula:**

- Usually iatrogenic.
- After drainage of abscess & appendectomy in presence of mass.
- Other specific causes as Crohn's disease.

- ▶ To avoid such complications, abdominal U/S is done.
- ▶ If mass is found → conservative treatment.

II. General Complications: (*rare*)

1- **Septicemia, toxemia, bacteremia or pyemia.**

2- **Portal pyemia (Pylephlebitis):**

- It is a grave condition (*rare*).
- **Cause:**
 - Suppurative thrombophlebitis of portal venous system.
- **Clinical Picture:**
 - Chills, high fever, low grade jaundice and later hepatic abscess.

Clinical Picture

A- **Typical Clinical Picture**

Proper history taking and physical examination are the backbone of diagnosis

➤ **Symptoms**

1- **Acute Pain that shifts (presenting symptom):**

- It starts periumbilical and is ill defined colicky (visceral pain).
- It is due to distension of the appendix.
- ▶ **Within 6 -10 hours the pain shifts to right iliac fossa:**
 - Due to irritation of parietal peritoneum.
 - Becomes sharper (Somatic pain).
 - Aggravated by movement or cough.

2- **Anorexia & Nausea:**

- Usually present.

3- **Vomiting :**

- 75% of patients.
- More severe in obstructive type.
- Not usually repeated.
- It is always preceded by pain.

4- **Constipation:**

- Usually present.
- Diarrhea may present early in the course of appendicitis (1 or 2 motions) or in case of pelvic or retroileal appendicitis

- A- Typical C/P including the special signs.
- B- Atypical forms.
- C- Picture of complications.

- ▶ If the patient is asked to cough, the pain becomes sharp and localized to the site of appendix.
- ▶ Hard to diagnose if patient < 5 years.

General Examination

1. Mild fever $< 38^{\circ}\text{C}$ (If higher indicates complications or suggest other causes of acute abdomen).
2. Tachycardia not prominent (< 100): (Proportionate to fever)
(If higher indicates complications)

Local Examination

► The signs of appendicitis are more evident on McBurney's point

1. Inspection:

- ↓ Abdominal movement with respiration → indicates peritonitis.
- **Rigidity** on the right iliac fossa (involuntary muscle spasm)

2. Palpation:

- Localized **tenderness** in right iliac fossa.
- **Rebound tenderness** on the right iliac fossa.
- **Guarding** on the right iliac fossa (voluntary muscle spasm).

3. Auscultation:

- There may be **diminished intestinal movement**.

4. PV & PR:

- To detect **pelvic appendix** & exclude gynecological causes.

► In obstructive type:

- **Severe colicky** Pain with **rapid shift**.
- **More severe vomiting**.
- **Marked** fever & tachycardia.
- **Marked** tenderness & rigidity.

Special Signs

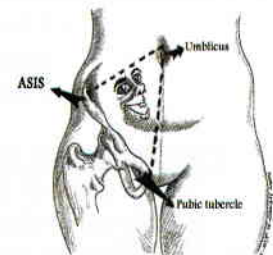
1. Rovsing's sign: (***crossed tenderness***)

- Pressure on left iliac fossa → pain on right iliac fossa due to:
 - Shifting of gases from the pelvic colon to the caecum.



2. Hyperaesthesia of triangle of Sheren:

- This is due to irritation of parietal peritoneum.
- It disappears after perforation.
- It is from:
 - Umbilicus.
 - Symphysis pubis.
 - Anterior superior iliac spine.



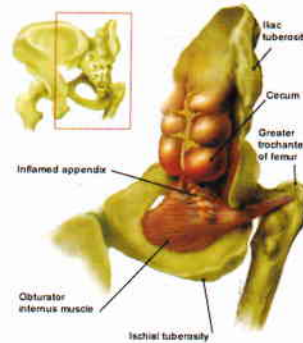
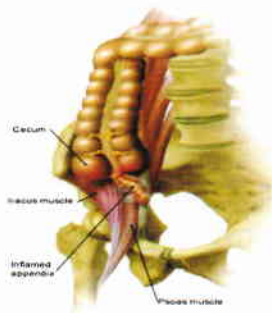
3. Obturator sign (Zachary Cope): (***in pelvic type***)

- Pain on internal rotation of flexed right thigh due to irritation of the obturator internus muscle by pelvic appendicitis.



4. Psoas sign: (in retrocaecal type)

- Pain on extension of the right thigh due to psoas spasm as a result of irritation by retrocaecal appendicitis.



B- Atypical Forms

1. Long Retrocaecal appendix (74%):

- Pain & rigidity are minimal and only on McBurney's point due to presence of caecum over the appendix.
- The appendix might touch the psoas muscle producing flexion deformity & **+ve psoas sign**.
- The appendix might touch the ureter producing ureteric colic.

2. Pelvic appendix (21%):

- It might touch the following structures:
 - Obturator Internus muscle → lateral rotation of the hip & **+ve obturator sign**.
 - Bladder → dysuria.
 - Rectum → dysentery & diarrhea.
- The appendix might be felt in P/R on the right side & not on the left side.
- Usually no tenderness at McBurney's point sometimes deep tenderness can be made just above symphysis pubis.

3. Paracaecal appendix (2%):

- Classic presentation.

4. Postileal appendix (0.5%) [*Missed Appendix*]:

- It touches the ileum producing **diarrhea** (enteritis) then constipation (paralytic ileus).
- Tenderness if any may present to the right of the umbilicus.

➤ Postileal type is considered the most dangerous type ☑:

1. No shift of pain which is misdiagnosed as gastroenteritis → "**Missed appendix**"
2. No omental localization → **Peritonitis**
3. Affection of iliocaecal vein → **Portal pyemia**
4. Affection of appendicular artery → **gangrene**

5. Subhepatic appendix:

- Failure of descent of the caecum → signs are higher than McBurney's point.
- It simulates acute cholecystitis but:
 - The patient is young.
 - Hyperaesthesia in the triangle of Sheren.
 - Pain is not referred to back or shoulder.

6. Appendicitis in pregnancy:

- No localization as omentum is elevated.
- The pain displaces upwards as the pregnancy progresses.
- Should be differentiated from pyelitis.
- Effect:
 - Imperforated → Premature labor (5%).
 - Perforated → Abortion or premature labor (20%).

7. Appendicitis in children:

- Serious condition → 80% would have perforated appendicitis due to:
 - Misdiagnosed as gastroenteritis due to vomiting.
 - Difficult examination.
 - Immature omentum.
 - Difficult localization.

8. Appendicitis in old age:

- Usually there is slight tenderness & rigidity with high risk of perforation due to natural weakness of the immune system.
- Cancer caecum must be excluded.

9. Appendicitis in obese persons:

- Local signs may be difficult to elicit.
- Midline abdominal incisions may be required due to delayed diagnosis and technical difficulties.

C- Picture of Complications

1. Appendicular mass:

- History from 2-3 days (in non-obstructive).
- Temperature > 38°C, more tachycardia & vomiting.
- Ill defined fixed mass with muscle rigidity might be felt in the right iliac fossa (sometimes missed due to guarding).
- Mass might be felt under general anesthesia.



➤ Mass will not be formed in the following conditions [☑]:

1. Children not > 10 years of age as the omentum is not well developed.
2. Elderly as there is atherosclerosis of the appendicular artery & perforation is easier.
3. Pregnancy as the omentum is shifted upward.
4. Diabetics as the general condition is poor.
5. Intake of purgatives.

2. Appendicular abscess:

- History of appendicitis 5 days earlier.
- S & S of pus loculus formation (hectic fever & throbbing pain).
- ↑size of mass - ↑Gastric aspiration - ↑abdominal pain

3. Peritonitis:

- History suggestive of appendicitis.
- Fever over 38°C.
- S & S of diffuse peritonitis develop.

➤ Acute appendicitis diagnostic facts

- Diagnosis is essentially clinical.
- Acute pain that shifts.
- Anorexia is almost always present. If the patient feels hungry & can eat the diagnosis of acute appendicitis is in serious doubt.
- Pain precedes vomiting. If vomiting comes first consider the diagnosis is gastroenteritis.
- Localized pain especially in Rt iliac fossa. sometimes point of tenderness is low in pelvis or high in Rt hypochondrium (according to site of appendix)
- A normal leucocyte count doesn't exclude appendicitis but high count with shift to left support the clinical diagnosis.

Investigations

(It is a **clinical diagnosis** i.e. investigations are needed mainly to exclude other causes of acute abdomen)

1. Laboratory:

1. Leucocytic count:

- Moderate leucocytosis (10,000 – 16,000/ul), However normal WBCs count does not rule out appendicitis.

2. Urine analysis: To exclude UTI or urinary stones.

3. Pregnancy test: If ectopic pregnancy is suspected.

2. Radiological:

U/S:

- May diagnose appendicitis (dilated lumen, thick walled appendix) in few cases (sensitivity 75%, specificity 95%)
- Exclude other diseases (Ureteric stones, Gynecological problems, etc.).

3. Instrumental:

Laparoscopy:

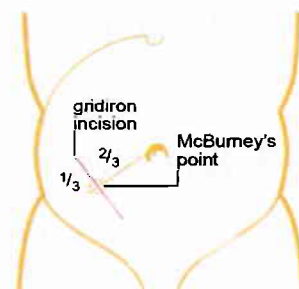
- Some surgeons have the policy to perform laparoscopy for females in the childbearing period with lower right abdominal pain.
- Advantages:
 - If the problem is medical (PID, Midovulatory pain); we protect the patients from hazards of surgery.
 - If the problem is surgical (Acute appendicitis, ovarian cyst); we can deal with it laparoscopically.

Treatment

1. Uncomplicated

Acute appendicitis.

- **Urgent appendectomy** through Gridiron incision at point of maximum tenderness (mostly at McBurney's point)
- The operation shouldn't be delayed specially in children, elderly, pregnant females & diabetics.



2. Complicated

1. Appendicular mass:

Conservative management (Oschner Sherren)

- This is due to marked adhesion so the operation is difficult & injury of the caecum might lead to fecal fistula.

➤ **What is Oschner Sherren's regimen?**

1. Semisitting position to relax the abdominal muscles & to make the pelvis more dependent.
2. Ryle if there is vomiting for 48 hours.
3. Intravenous fluids (until nausea subsides, then oral intake starts with fluids followed by gradual introduction of solid meals).
4. Intravenous antibiotics:
 - Ampicillin + Metronidazole + Aminoglycosides.
5. Monitoring of:
 - Vital signs.
 - Degree of tenderness
 - Vomiting
 - Size of the mass.

➤ **Results of conservative management:**

- 1- Mass↓, fever↓, pain↓ → appendectomy after 3 months.
- 2- Pain ↑ (throbbing), fever ↑ (hectic) → abscess.
- 3- Pain↓, fever↓, but mass persists → investigate as cancer caecum.
If regress → **appendectomy after 3 months** ☑.

➤ **How to know improvement of the mass?**

1. C/P:

- Symptoms: ↓ pain, ↓ vomiting, no constipation.
- Signs: ↓ abdominal signs, ↓ mass, ↓ fever & tachycardia.

2. Investigations:

- ↓ Leucocytosis in acute appendicitis → >11000 / cmm and if complicated >20000 / cmm.

➤ **When it is contraindicated to do Oschner Sherren's regimen?**

1. Patient presented with abscess.
2. Doubtful diagnosis.
3. Dangerous group (children >10 yrs ,elderly , pregnancy , DM)

2. Appendicular abscess: (complicated mass)

Drainage & Antibiotics

1. Muscle cutting incision
2. Extraperitoneal drainage
3. Drain is introduced
4. The appendix is not removed:
 - Pelvic abscess → Transanal drainage through anterior wall of the rectum.
 - Iliac abscess → iliac incision over point of maximum tenderness

In all cases → **Appendectomy after 3-6 months** ☑.

3. Diffuse Peritonitis:

1. Resuscitation & Monitoring.
2. Exploration.
3. Removal of the appendix.
4. Peritoneal toilet.

5. Three drains:

- Pelvic drain → most dependent while standing.
- Hepatorenal → most dependent while lying down.
- Paracaecal.

4. Early Interference:

- In children, elderly & pregnancy because there is no localization.

5. Portal Pyemia:

- Ligation of ileocolic vessel + antibiotics + TPN.

▶ Indications of Laparoscopic Appendectomy:

- Female in child bearing period → avoid postoperative extensive fibrosis and adhesions → may lead to infertility.

▶ Acute appendicitis is more dangerous than acute cholecystitis?

1. Gall bladder has dual blood supply.
2. Gall bladder wall is stretchable.
3. Gall bladder obstruction resolves on treatment.

▶ Acute appendicitis is less dangerous than Meckel's diverticulitis?

1. Thin wall of the diverticulum.
2. No localization of the inflammation.

Differential Diagnosis of Acute Abdomen

Group 1: Thoracic problems

1. Rt. Pneumonia:

- Marked chest symptoms, minimal abdominal tenderness and there is no rigidity.

2. Tonsillar tummy:

- Child with acute tonsillitis → swallows pus → abdominal colic.

3. Diaphragmatic pleurisy.4. Myocardial infarction.

Group 2: Upper Abdominal problems

1. Perforated Peptic Ulcer:

- History of dyspepsia is present.
- Plain X-Ray shows air under the diaphragm.

2. Acute Cholecystitis:

- Pain in the right hypochondrium
- Fever is higher.
- U/S will confirm the diagnosis.

3. Intestinal Obstruction:

- Repeated vomiting.
- Absolute constipation.
- Multiple fluid levels in X-Ray abdomen erect.

4. Cyclic vomiting of children : temp is normal , no tenderness

Group 3: Lower Abdominal Problems

1. Non-specific Mesenteric Lymphadenitis ☑:

- Common in children.
- There is shifting tenderness

2. Regional ileitis.

3. Deep iliac adenitis:

- Child with septic focus in LL → pain in iliac fossa, psoas spasm → flexion deformity, high fever and O/E → tender nodular fixed mass in iliac fossa very close to inguinal ligament.

4. Mickle's Diverticulitis.**5. Perforated ileal typhoid ulcer:**

- History of typhoid, tenderness all over the abdomen X-Ray → free gas in peritoneum (erect → gas under diaphragm).

6. Diverticulitis of a long sigmoid colon.**Group 4: Pelvic problems**

- In adult females careful history, examination and U/S are essential to detect DD.

1. Disturbed right ectopic pregnancy:

- History of amenorrhea.
- Shock.
- Tender cervix.
- Vaginal bleeding.

2. Acute salpingitis:

- Fever, vaginal discharge, tenderness often bilateral.

3. Midcyclic pain (Mittelschmerz)**4. Twisted right ovarian cyst from appendicular mass.****5. PID:**

- Vaginal discharge, bilateral pain, mass felt on PV

- ➔ Condition 1-3 can be highly suspected if a careful detailed menstrual history taken especially as regards the exact date of last menstrual period and any delay in the cycle
- ➔ All condition from 1- 5 differ from appendicitis in that
 - ✓ Pain & tenderness are often bilateral and more in suprapubic area
 - ✓ Associated with bleeding may have mild hypothermia , tachycardia , pallor & may be low blood pressure
 - ✓ Fever may be present with salpingitis
 - ✓ PV examination is very informative
 - ✓ U/S is of great diagnostic importance

Group 5: Urological problems**1. Right ureteric coli:**

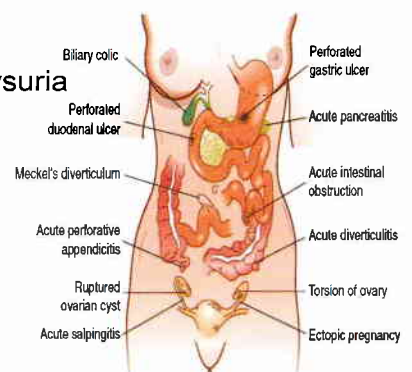
- Pain from loin → groin, pain does not increase with cough, patient writhing on himself while in appendicitis patient lies flat as movement increases pain.

2. Rt. Pyelonephritis:

- Fever 40°C + rigors, tender pain, dysuria
- Pain in loin radiate to groin rather than iliac fossa

Group 6: Neurological Problems**1. Disease of the spine:**

- Acute osteomyelitis & Pott's of dorsolumbar vertebrae.

2. Herpes Zoster in 10th, 11th, 12th thoracic nerves.**Others**

1. Diabetic abdomen.
2. FMF.

➔ **The commonest DD of acute appendicitis :**

- 1- Right tubo-ovarian causes
- 2- Gastro enteritis
- 3- Mesenteric adenitis (yersinia)
- 4- Meckel's diverticulum
- 5- Right uretric stone

DD of an appendicular mass

- Carcinoma of the caecum : associated with anemia and weakness and the mass is usually hard and not tender
- Crohn's disease
- Hyperplastic ileocaecal tubreculosis.
- Rt iliac lymphadenitis

- ▶ **McBurney's point** is the surface anatomy of the base of the appendix, which is the point of maximal tenderness in the Rt. Iliac fossa in appendicitis.
- ▶ Open in front of the base as it lies in a common fixed position while the apex has variable positions.
- ▶ The base could change its position in subhepatic appendix or situs inversus totalis which is rare.
- ▶ Bleeding during incision of McBurney's point is usually from superficial circumflex iliac vessels.
- ▶ Bleeding during muscle cutting is from deep circumflex iliac vessels.

Tumors of the Appendix

Carcinoid (Argentafinoma)

- ▶ **Path.:** Kulchitsky cells, tip of the appendix(70%)
- ▶ **C/P:** appendicitis ± carcinoid \$
- ▶ **Invest.:** 5-HIAA in urine, CT scan
- ▶ **TTT:** Appendectomy (< 2cm)
RT hemicolectomy (> 2cm)

▶ The commonest type

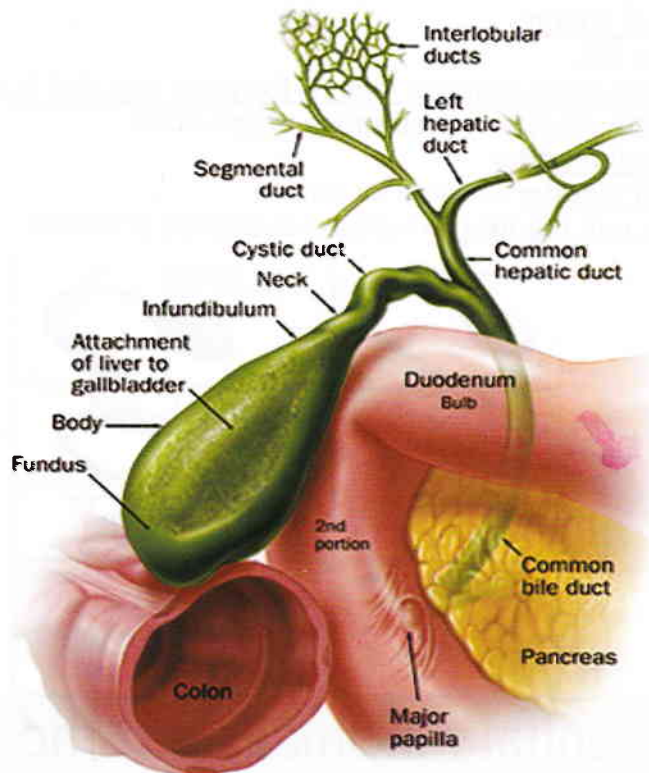
- A. **Origin:** Intramural nerve plexus in the base of the crypts of Lieberkuhn = **Kulchitsky cells**☐.
- B. **Site:**
- GIT → **appendix (70%)**☐ (**in the tip**), ileum (20%).
 - Outside GIT → testes, ovaries, bronchi.
 - 30% are multicentric.
- C. **C/P:**
- **Local:**
 - Acute or chronic appendicitis due to occlusion of the lumen → mass usually discovered intraoperative.
 - **Secondaries:** **Carcinoid \$**
 - Flushing, diarrhea, bronchospasm, ↑ ABP, Rt. sided valvular endocardial fibrosis
- D. **Investigations:**
1. 5-HIAA in urine.
 2. CT scan.
 3. Barium small bowel series: show teathering of distal ileum (not the tumor itself).
- E. **TTT**☐:
- Appendectomy if < 2 cm.
 - Right hemicolectomy if > 2 cm, careful inspection for synchronous lesions should be done.
 - If in the ileum → segmental resection of small bowel, enblock mesenteric resection & 1ry anastomosis.
 - Solitary or isolated liver, metastasis are resected if possible.

Adenocarcinoma

- It secretes mucin & can cause pseudomyxoma peritonii.
- **TTT**☐: Rt. Hemicolectomy.

- ▶ Both tumors might simulate appendicitis.
- ▶ That is why pathological investigations after appendectomy are important.

CHAPETR 7



THE GALL BLADDER

Congenital Anomalies

- Congenital anomalies of the biliary tract are present in 10% of autopsies.

Congenital Anomalies of the Gall Bladder

1. Anomalies of number:

- Congenital absence (0.03% of cases).
- Double gall bladder with single duct or double ducts (1 in 4000).

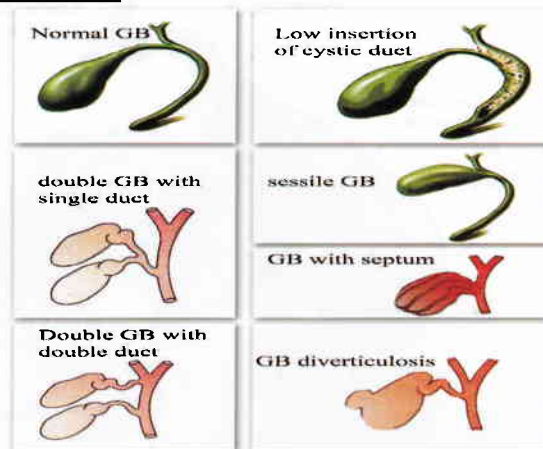
2. Anomalies of shape:

- Septate GB.
 - The most common form is: Phrygian cap (2-6 %): kinking of the fundus of the GB which may predispose to gall stone.

3. Left sided gall bladder

4. Floating gall bladder → liable for torsion

5. Intrahepatic gall bladder → increase incidence of stones



Congenital Anomalies of the Bile Ducts

1. Biliary atresia (see later).

2. Choledochal cyst (see later).

3. Hepatic duct anomalies:

- Accessory hepatic duct may enter into cystic duct or into the neck of the GB (during cholecystectomy [if they are present] may drain bile in the peritoneum {G. peritonitis}, so after cholecystectomy we put a drain until fibrosis).
- Rt., Lt. or both hepatic ducts may enter the G.B. Their ligation during cholecystectomy could result in surgical catastrophe.

4. Cystic duct anomalies:

- Low insertion of the cystic duct → post cholecystectomy syndrome.
- Sessile GB (no cystic duct) → injury of CBD during cholecystectomy.
- Cystic duct may enter RT.hepatic duct → mistaken for cystic duct and ligated.

Congenital Anomalies of the Blood Supply

A- Accessory cystic artery (the most common anomaly).

Biliary Atresia

(Neonatal obstructive jaundice)

Incidence

- 1 / 10,000 live births.

Etiology

- Unknown, may be the result of inflammatory process

Types

- 1- Correctable type (10%) : there is a patent portion of the extrahepatic duct which communicate with intrahepatic ducts
- 2- Non correctable type (90%) : the extrahepatic portion is occluded

Clinical Picture (Neonatal obstructive jaundice)

- Jaundice is present since birth[□] but is not marked until after 1st several weeks.
- Olive green jaundice, dark urine & clay colored stools).
- Distended abdomen by:
 - Hepatomegaly, ascites and splenomegaly (late).

Complications

- If untreated → biliary cirrhosis & death.

DD Causes of neonatal jaundice

- A. Physiological jaundice.
- B. Hemolytic diseases.
- C. Obstructive jaundice: e.g. inspissated bile syndrome, choledochal cyst, α -1 antitrypsin deficiency
- D. Hepatocellular jaundice:
 - Neonatal hepatitis. *(the most important item in DD)*
 - Viral infections.
 - Metabolic defects.

Investigations

1. Laboratory:

- **LFTs:** cholestatic pattern
 - ↑ serum bilirubin (direct fraction is at least ½ of the total).
 - ↑ alkaline phosphatase.

2. Radiological:

- **U/S:** hepatomegaly.
- **HIDA scan**[□]: non-visualization of the duodenum (no passage of bile and isotope to the duodenum).
- **ERCP**

3. Instrumental:

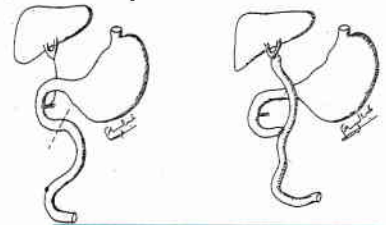
- **Liver biopsy:**
 - In intrahepatic atresia → no bile ducts.
 - In extrahepatic atresia → dilated intrahepatic bile ducts.
- **Kidney functions tests**

- ▶ **Types:** correctable (10%), non-correctable (90%)
- ▶ **C/P:** Neonatal obstructive jaundice since birth, hepatomegaly, ascites
- ▶ **Comp.:** biliary cirrhosis & death
- ▶ **Invest.:** as OJ + HIDA scan → NO visualization of the duodenum
- ▶ **TTT:** - Intrahepatic: transplantation
- Extrahepatic: Kasai operation

Treatment

Should be done before 2 months or around it

- **Intrahepatic** → Liver transplantation.
- **Extrahepatic** → (Removal of extrahepatic stenosis & its replacement by small intestine.
 - 1- If there is a patent segment of the proximal bile duct (10% of cases): Roux-en-Y operation is done with 75 % success rate (hepatico-jejunostomy).
 - 2- If no patent segment (90% of cases): **Kasai operation** (porto-enterostomy) in which excision of bile ducts up to liver capsule is performed and Roux-en-Y loops of jejunum is anastomosed to expose area of liver capsule, unsuccessful after 3 months of age.



Roux-en-Y operation

Choledochal Cyst (Obstructive jaundice in childhood)

Definition

- Congenital cystic dilatation of intra and / or extrahepatic biliary system.

Incidence

- There is a female predominance .

Etiology

(Unknown)

Clinical Picture

(Childhood obstructive jaundice)

The classic triad is jaundice, pain & right hypochondrial mass.

- 1- **Stasis:**
 - Cholangitis (jaundice, pain and pyrexia).
 - Gall stone.
- 2- **Swelling:**
 - In the Rt. hypochondrium.
- 3- **Rupture:**
 - Biliary peritonitis.

The majority of cases are diagnosed in childhood before 10 years of age

Complications

- **Cholangiocarcinoma**.
- Biliary cirrhosis.

Investigations

- See biliary atresia

Treatment

- Excision of the cyst + choledochojejunostomy (Roux-en-Y with CBD).

- ▶ **Def.:** cong. cystic dilatation of biliary system
- ▶ **C/P:** ♀ child with jaundice, pain, swelling in the Rt. hypochondrium
- ▶ **Comp.:** cholangiocarcinoma, biliary cirrhosis
- ▶ **Invest.:** as biliary atresia
- ▶ **TTT:** Excision + Choledochojejunostomy

Investigations of Biliary System

1- Plain X-Ray:

- Detects gall stones in 10-15 % of cases.
- Hydrops of GB and carcinoma of GB may appear as right upper quadrant (RUQ) mass on plain film.
- Porcelain GB: calcified GB: (associated with carcinoma in 25% of cases).
- Air in GB (emphysematous cholecystitis) or fistula with duodenum or in biliary system (after choledochoduodenostomy or sphincterotomy operations).



Multiple calcified faceted gallstones

2- Abdominal U/S:

The 1st investigation done in jaundice. i.e. 1st screening modality.

▪ Advantages:

- Easy, non-invasive and inexpensive, quick to perform.
- Safe in pregnancy and pediatrics (use non-ionizing radiation).
- Adjacent upper abdominal structure can be imaged at same time.
- Can be done in acute inflammation & jaundice.

▪ Disadvantages:

- Operator dependant.
- Suboptimal in:
 - Excessive body fat
 - Excessive bowel gas

▪ It can detect:

- a- GB stones in 98%[□] of cases (very accurate)
- b- CBD stones in 70%[□] of cases (less accurate)
- c- Thickening of the GB wall (GB may be shrunken, fibrotic or edematous).
- d- Masses in porta hepatis or head of pancreas.
- e- Dilatation of extra and intra hepatic biliary ducts in patients with obstructive jaundice.
- f- Size of the GB.



Solitary stone Gall bladder

- **The normal diameter of CHD is 4-6 mm or less, greater than 8 mm[□] is considered abnormal.**
- **Some patients (e.g. sclerosing cholangitis) may have significant biliary obstruction without dilated ducts.**

3- HIDA scan (hydroxy imedinodiacetic acid):

▪ Method:

- Tc⁹⁹ labelled derivative of HIDA is injected IV and rapidly taken and excreted by the retroendothelial cells of the liver to visualize the biliary tree and GB (within 30 minutes of isotope injection in 90% of cases).

▪ Uses[□]:

- 1- Diagnosis of acute cholecystitis (non-visualization of the GB indicates obstruction of its neck)
- 2- Diagnosis of congenital biliary atresia & neonatal hepatitis (no uptake).
- 3- Diagnosis of biliary enteric anastomosis (better than endoscopy because the anatomy is disturbed).
- 4- Diagnosis of biliary leak (confirms presence, site and quantity of leak).

4- ERCP: endoscopic retrograde cholangio-pancreatography.

- **Method:**

1. An endoscope is introduced to the 2nd part of the duodenum.
2. A cannula is introduced by side view endoscopy for visualization of the ampulla.
3. Injection of radio-opaque material followed by X-ray films.

- **Diagnostic value:**

- 1- Visualization of stone which may be:
 - Floating → filling defect or impacted.
 - Single or multiple.
- 2- Visualization of extrahepatic & intrahepatic biliary system.
- 3- Sample of bile or pancreatic juice (could be aspirated & sent for cytological & microbiological examination).
- 4- Visualization & biopsy of tumors that invade the duodenum (tumors involving lower end of CBD).
- 5- Detection of operative injuries of biliary system.
- 6- Detection of missed calculi in the CBD after cholecystectomy.
- 7- Visualization of pancreatic duct in chronic pancreatitis or pancreatic pseudocyst.
- 8- Is not useful in acute cholecystitis and can't detect gall stones in most cases.

- **Therapeutic value:**

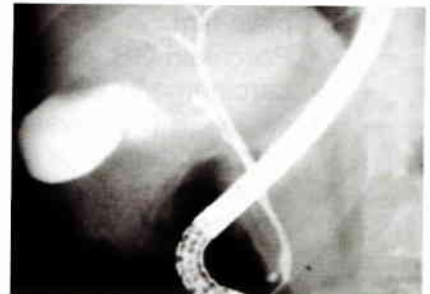
- 1- Endoscopic sphincterotomy + extraction[☑] of the stone by dormia basket.
- 2- Insertion of a stent[☑] to drain malignant obstruction of the duct.
- 3- Stricture dilatation[☑].

- **Complications:**

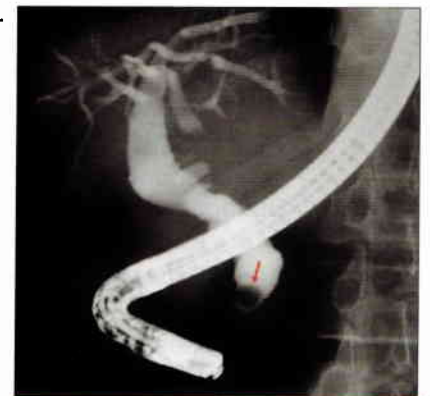
1. Ascending cholangitis (**COMMONEST**[☑]) or even septicemia.
2. Bleeding → hemobilia due to sphincterotomy.
3. Acute pancreatitis or duodenal perforation.
4. Rupture or perforation of CBD.

- **Precautions:**

Prophylactic antibiotics (3rd generation Cephalosporins) → (more concentration in bile).



Normal ERCP



ERCP common duct stones

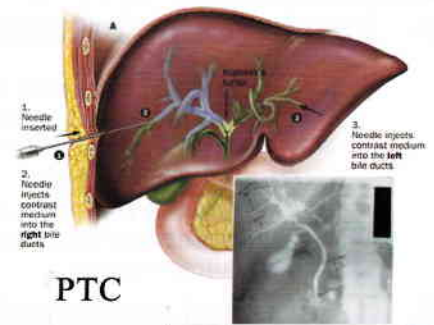
With the widespread availability of US and the new comer MRCP, ERCP becomes a therapeutic rather than a diagnostic technique.

5- PTC:▪ **Method:**

1. Under local anaesthesia and U/S intrahepatic biliary passage is punctured in the midaxillary line through 8th intercostal space by Chiba needle Catheter is passed (better to be done on dilated intra hepatic biliary ducts).
2. Injection of radio-opaque material followed by X-Ray films.
3. The procedure is done under fluoroscopic guidance and mild sedation. Pre-procedure antibiotic coverage is recommended.

▪ **Diagnostic value:(high accuracy)**

- 1- Particularly indicated to diagnose high obstruction of bile ducts.
- 2- Visualization of the extrahepatic & intrahepatic biliary system.
- 3- It is done if the ERCP fails to give enough data about the obstructing agent.



Normal PTC



PTC stones in CBD

Distal lesion of biliary tract e.g. CBD stones, cancer head of pancreas are better seen with ERCP, while proximal lesions e.g. intrahepatic ducts are better seen with PTC than ERCP (because retrograde flow into intrahepatic ducts may be blocked).

▪ **Complications:**

- 1- Liver injury and hemorrhage → hemobilia or hemoperitoneum..
- 2- Sepsis.
- 3- Biliary peritonitis.

▪ **Prerequisites:**

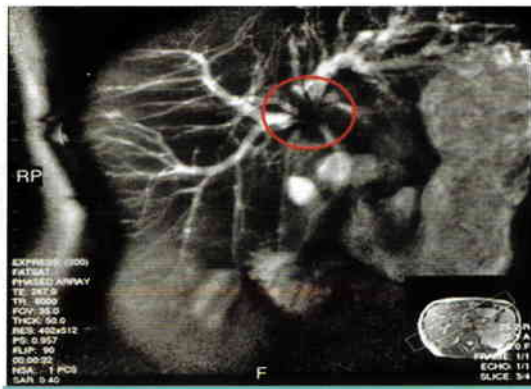
- PT and concentration should be done before the procedure if prolonged vitamin K is given I.V for a few days before the procedure.
- Dilated intrahepatic biliary radicles as seen on U/S.

6- MRCP (Magnetic Resonance Cholangiopancreatography):▪ **Anvantages:**

- Non-invasive without need to inject contrast material.



2 CBD stones and dilated CBD



Hilar cholangiocarcinoma

7- Intraoperative Investigations:

a. Choledoscopy:

- Is used to visualize the bile duct.
- Its use makes the incidence of missed stones ZERO in treatment of calcular obstructive jaundice.

b. Cholangiography.

- While the patient is tilted, head down 20 degrees and taking care not to introduce air bubbles into biliary system as it appears as stones giving false positive results.

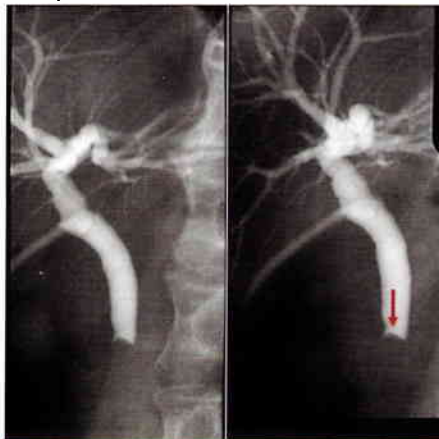
8- T-tube cholangiography:

a. Intraoperative to detect any missed stones

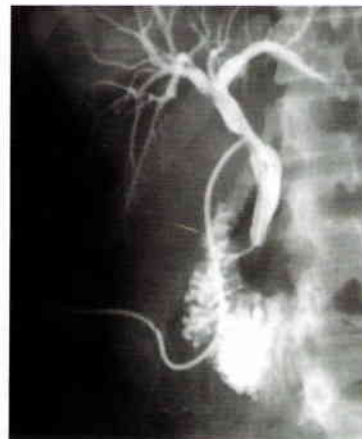
- A T-tube is usually inserted into the CBD following CBD exploration.
- ↓incidence of missed stone from 10-20% to 0%

b. Postoperative:

- If intraoperative imaging is not available, but in this case there is a high incidence of missed stones.
- Cholangiography prior to T-tube removal is usually performed 7-10 days postoperative to check for missed stone.
- If retained stones are identified the mature T-tube tract (after 6 weeks) can be used as a route for non-operative stone extraction.
- Other therapeutic maneuvers such as balloon dilatation of bile duct strictures or stent placement can be done via the T-tube tract.



Radiologic extraction of retained common duct stone



Normal T-tube cholangiography

9- Oral Cholecystography: (not done now)

▪ Value:

- Oral Cholecystography visualizes the GB only (not the biliary tree).
- Shows 90% of the GB stones as a filling defect.

▪ Indication: the only indication is:

- Chronic Non-calicular Cholecystitis: to detect that it is non-functioning

▪ Contraindications:

- ✓ During acute cholecystitis.
- ✓ Jaundice, liver failure or renal failure.

- **Technique:**

- ✓ Give the fasting patient 6 tablets of telepaque (tetra-iodo-phenolphthalein) 12 hours before taking X-Ray film.
- ✓ The patient is then given a fatty meal & another film is taken after ½ hour.
- ✓ Normally → gall bladder well opacified, then after a fatty meal GB will contract, so its size will shrink to ⅓ of its original size.

OCG was the standard investigation for GB until the advent of US in mid 1970's. More recently it has been used as a technique for assessing gall stones (size, number, cystic duct patency) in patients being considered for lithotripsy and other non-surgical therapies for gall stones.

- **Non visualization of GB may be due to:**

1. **Failure of the dye to reach liver:**

- Patient did not take the dye.
- Vomiting of the dye.

2. **Failure of the liver to excrete the dye:**

- Liver dysfunction.

3. **Failure of the dye to reach GB:**

- Obstruction of cystic duct.
- Absence of the GB (congenital or surgically removed).

4. **Failure of the GB to concentrate the dye:**

- Chronic cholecystitis.

- **Evidences of chronic cholecystitis in cholecystography:**

1. **F**ibrosis → distorted shape of GB.
2. **F**illing defects due to radiolucent stones.
3. **F**aint concentration of the dye.



Cholecystography showing multiple GB stones

Gall Bladder Stones

Incidence

- 20% of females more than 40 years (female, fatty, fertile, forty or fifty & flatulent)✓.
- More common in western countries.
- Female: male → 3:1, but this female predominance is not present after 60 years of age and in pigment stones.
- There is also familial incidence.
- The incidence increase with age

- ▶ It is common in 5F (Female, Fatty, Fertile, Forty, Flatulent)
- ▶ **Etiology:** metabolic causes (bile salt / cholesterol ratio), infection or stasis of bile
- ▶ **Types:** cholesterol, mixed, pigment
- ▶ **C/P:** asymptomatic (70%), biliary colic (15%) biliary dyspepsia ± +ve murphy's sign
- ▶ **Comp.:** OMUMI
- ▶ **Invest.:** U/S, x-ray, LFTs, KFTs
- ▶ **TTT:** - Asymptomatic: no treatment
- Symptomatic : cholecystectomy
- TTT of complications

Causes

A- Cholesterol and mixed gall stones:

1- Disturbed bile salts cholesterol ratio:

- A certain ratio (25 : 1) between bile salts and phospholipids on one hand and cholesterol on the other hand has to be maintained to keep cholesterol in solution.
- Any lowering of this ratio can lead to supersaturated bile (lithogenic bile) with consequent cholesterol precipitation.
- The following factors may disturb this ratio :
 - Reduced bile salt pool
 - Malabsorption of bile salts in the terminal ileum in crohn's disease, small bowel resection
 - Diminished hepatic synthesis in liver disease
 - Estrogens reduce the concentration of bile salts in bile
 - Increase cholesterol synthesis in obesity , high dietary fat and high caloric diet

2- Stasis of bile:

- Progesterone causes relaxation and impaired emptying of the gall bladder. estrogen , oral contraceptives and repeated pregnancy
- Following truncal vagotomy due to denervation of gall bladder
- Diabetes mellitus
- Obesity
- Long term parenteral nutrition

B- Pigment stones:

- Haemolytic anaemias
- Liver cirrhosis : due to decreased secretion of bile acids by the cirrhotic liver leading to diminished solubility of any unconjugated bilirubin
- Infection : plays a role in the formation of brown pigment stones. Infection by some strains of E.coli leads to the production of B-glucuronidase enzyme which hydrolyses bilirubin glucuronide into the insoluble bilirubin which precipitate as calcium bilirubinate.

Types

	Cholesterol	Mixed	Black pigment	Brown pigment
Incidence	8%	80% [☑]	Pt with hemolytic anemia & cirrhosis	
Constituents	Pure cholesterol	Cholesterol(60%), CaCO ₃ , Ca bilirubinate Bile salts, bile pigments Phospholipids	Calcium bilirubinate	Major → calcium bilirubinate. Others → calcium malmitate and cholesterol
Number	- Usually single [☑] (cholesterol solitaire) - Sometimes multiple	Multiple	multiplable	multiaple
Size	Large >2.5cm	0.5-2.5cm. Arranged in groups each group contains equal sized stones & represents an attack of infection.	<2.5 cm	<2.5 cm
Shape	Rounded or oval	Faceted	Multiple specula	laminated
Surface	Mamillated	Smooth		
Color	Yellow	Yellowish	Tarry black	brown
Consistency	Hard & floats	Hard & sinks		
Cut section	No nucleus Radiating	Nucleated + laminated		
X-Ray	Radiolucent [☑]	Radio-opaque [☑] in 15% of cases	Radio opaque in 50 % cases	Radio opaque



Mixed gall stones



Clinical Picture

- **Type of patient:**
6F (Female, Forty or Fifty, Fatty, Fertile, Flatulent)
- **Asymptomatic:** (Most common ☒ 70%)
 - Discovered accidentally by plain X-Ray or abdominal U/S done for another problem.
- **Symptomatic gall stones:**
 - **Biliary colic:** (Sudden onset of colic in the rt. hypochondrium) (about 15% of cases)
 - Radiates to: **Rt. shoulder, the back** & inferior angle of the rt. scapula along intercostal, phrenic & suprascapular N (C₃, C₄) and usually starts at night.
 - Severe attacks may be accompanied by nausea & vomiting.
 - If persists → acute cholecystitis. (less than 5-6 hrs)
 - Precipitated by: fatty meals
 - Relieved by: antispasmodics.
 - Usually recurrent attacks.
 - Reflex symptoms: reflex retrosternal pain usually diagnosed as anginal pain, and actual ECG changes may occur.
 - **Biliary dyspepsia:** (abdominal distention, excessive eructation after fatty meals)
- **Complicated gall stones e.g.:**
Obstructive jaundice: jaundice, itching, dark urine, clay stool.
- **On examination:** tenderness in the right hypochondrium, and Murphy's sign may be +ve.

- It is of interest that the patient may have several episodes of biliary colics over period of a few weeks and then no more troubles for some months.

Complications

OMUMI

1. Obstruction of:

1. **Hartmann's pouch or cystic duct** → biliary colic, acute cholecystitis, mucocele ☒, empyema.
2. **CBD** → obstructive jaundice, white bile (**bad prognostic sign**), hydrohepatosis, ascending cholangitis, biliary cirrhosis.
3. **Ampulla of Vater** → pancreatitis (due to activation of pancreatic juice inside pancreas by regurgitated bile).



Empyema gall bladder

2. Migration:

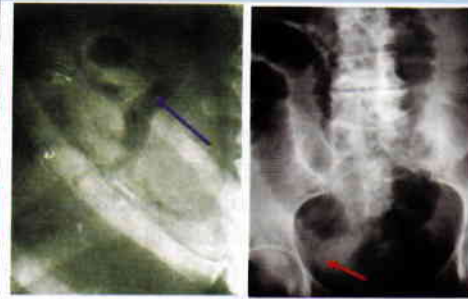
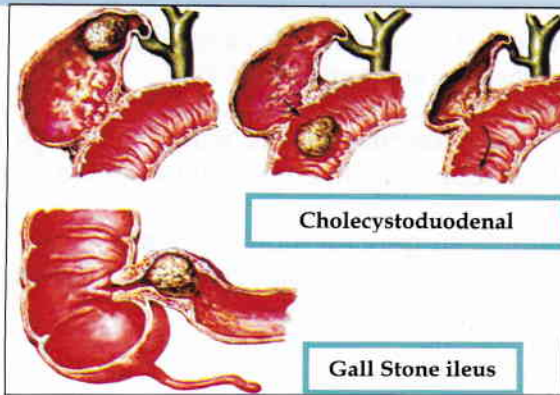
a. **Through cystic duct:** biliary colics & transient jaundice.

b. **Through fistula:**

i- **Cholecysto-choledochal fistula:**

Mirizzi Syndrome ☒.

- **Type I** → Stone is impacted in Hartmann's pouch pressing on CBD → obstructive jaundice.
- **Type II** → Prolonged pressure → fistula between GB and CBD..
- **Treatment:**
 - ERCP sphincterotomy → pulling of stone by dormia basket.
 - Then, cholecystectomy & exploration of CBD closing the fistula.



ii- Cholecysto-duodenal fistula:

- Gall stone ileus[☐] (due to obstruction at terminal ileum)
- Usually in old females
- Stop 2 feet from ileocecal valve
- Plain x-ray abdomen → gases in the biliary tree[☐] (aerobilia), multiple air fluid level.
- Treatment: (2 stage operation):
 - Remove the stone (enterostomy) or crush it.
 - Cholecystectomy & repair fistula.

iii-Cholecysto-colic fistula:

- Passes with stools.

3. Infection: cholecystitis, ascending cholangitis, pancreatitis.

4. Ulceration: hemobilia (very rare).

5. Metaplasia: squamous metaplasia followed by carcinoma[☐] (very rare).

DD

Of dyspepsia (will be discussed later)

Investigations

I. For diagnosis

- Abdominal U/S: (inv. Of choice[☐])
 - Detects size & thickness of GB & presence of the stone (in 98% of cases).
 - Detects diameter of CBD and any intrahepatic biliary dilatation.
- Plain X-Ray:
 - Only 10-15%[☐] are radio-opaque.
 - They are multiple, faceted signet ring.
 - They lie anterior to spine in lateral view
- Oral cholecystography: Rarely performed now.



Plain X-Ray showing multiple stones

II. For complications:

- LFTs: usually normal
 - Bilirubin, ALP, gamma GT.
 - (If high direct bilirubin & ALP → Stone in CBD → ERCP is needed)
 - PT, albumin, SGOT, SGPT.
- KFTs:
 - Creatinine, BUN.

Treatment

CBD stones are found in 15% of patients undergoing cholecystectomy (without preoperative ERCP).

- **Asymptomatic Gall Stones:** *No treatment (wait & watch[☑]) except in:*
 - Diabetic patient (controversial)(because if acute cholecystitis occurs → gangrene)
 - Congenital hemolytic anemia
 - Patients who will undergo Bariatric surgery for morbid obesity.
 - Young fit patient as life span is long and there high chance for development of complications.
 - Porcelain gall bladder → precancerous
- **Symptomatic Gall Stones:** *Cholecystectomy is the standard treatment[☑]*
- **Treatment of Complicated GB:**
 1. **Acute Cholecystitis:**
 - Patient > 48 hours) → conservative treatment then cholecystectomy (recently done laparoscopically).
 - Patient < 48 hours → urgent cholecystectomy.
 2. **Acute Pancreatitis:**
 - Conservative treatment.
 3. **Obstructive Jaundice:**
 - First we do ERCP:
 - If succeeds we do laparoscopic cholecystectomy.
 - If fails we do open cholecystectomy & explore CBD.
 4. **Gall Stone Ileus:**
 - Relief of intestinal obstruction → Resuscitation & Monitoring.
 - Deal with the stone:
 - Crushing of the stone.
 - Milking to pass ileocecal valve.
 - Removal by enterostomy.
 - Resection & anastomosis.



Acute Cholecystitis

Etiology

- **Calcular (98%):** 2ry to stones in the cystic duct or Hartmann's pouch.
- **Non-calicular (2%).**

A- Acute Calcular Cholecystitis

Microorganisms

- Usually by gram -ve e.g. E.coli, Proteus & Klebsiella.
- May be Staph., Strept.,
- Emphysematous gall bladder caused by Clostridium welchii or salmonella (Typhoid).

- ▶ the commonest type of acute cholecystitis
- ▶ **Etiology:** E.coli + direct or blood spread
- ▶ **C/P:** a- 6F, FAHM, abdominal pain, N.&V.
b- O/E: signs of acute abdomen (max. at Rt. Hypochondrium, Boas' sign, GB mass)
- ▶ **Comp.:** general, local (empyema, spread,...)
- ▶ **Invest.:** U/S (inv. of choice), HIDA
- ▶ **TTT:** -Early(<48h.): urgent cholecystectomy
-Late(>48h.) conservative TTT

Routes of Infection

- Direct extension → ascending from duodenum.
- Blood spread → from chronic septic focus (via cystic artery).

Pathogenesis

- Obstruction of cystic duct or neck of GB by stone → stasis → infection.

Pathology

Types

- Catarrhal, suppurative or gangrenous.

Macroscopic Picture

❖ Gall bladder:

- **Shape:** distended.
- **Lumen:** may contain stones.
- **Wall:**
 - **All layers:** edematous & hyperemic ± multiple micro-abscesses.
 - **Mucosa:** Congested ± patchy ulceration.
 - **Serosa:** Loss of peritoneal luster and covered by fibrinous deposits
 - As a defensive mechanism, the greater omentum, the duodenum and colon become adherent to the gall bladder to localize the infection.

❖ Cystic lymph nodes of Lund: Enlarged & elastic.

Clinical Picture

Characteristic **6F patient** with **past history** of gall stones (biliary colic, biliary dyspepsia)

Symptoms

- **General:**
FAHM (fever is high).
- **Local:**
 1. **Pain:**
 - At first: It is diffuse colicky upper abdominal pain.
 - Then it becomes dull-aching, localized to right upper quadrant.
 - May be referred to: to the Rt shoulder due to irritation of under surface of the diaphragm (supplied by sensory fibers of phrenic n.).
 2. Usually there is nausea, sometimes vomiting.

Examination

General:

- A characteristic patient:
(6F: Female, Forty or Fifty, Fatty, Flatulent, Fertile)
- High temperature (fever) & tachycardia.
- Jaundice rarely large stone may be impacted in Hartman's pouch and compress bile duct (MIRIZZI syndrome)

Local (maximum on the Rt. hypochondrium):

- Signs of acute abdomen → marked at Rt. Hypochondrium.

- ✓ Inspection: rigidity.
- ✓ Palpation: tenderness, guarding, rebound tenderness.
- ✓ Percussion: -ve.
- ✓ Auscultation: decreased intestinal sound.
- ✓ DRE: -ve

Murphy's sign may be detected

Special signs:

- ✓ Boas' sign: An area of hyperesthesia between Rt. 9th & 11th ribs posteriorly.
- ✓ GB mass: difficult to palpate due to overlying tenderness and rigidity.

Consequences

- Resolution: in most cases, with treatment, the stone dislodges and the obstruction is relieved with gradual resolution of inflammation.

Complications:

General

- Toxemia, septicemia, pyemia.

Local (Presented by throbbing pain & hectic fever)

1. Empyema and mucocele.
2. Chronic change:
 - Due to: bad general condition, inadequate treatment, or **persistence of the predisposing factors (stones)**.
 - Clinical picture: recurrent attacks of biliary pain and biliary dyspepsia.
3. Spread of infection:
 - Cholangitis, (Charcot's triad), cholangiohepatitis.
 - Pancreatitis: fever & pain radiating to back decreasing by leaning forward, O / E → acute abdomen.
4. Perforation (Rupture): Rare.
 - Sites:
 - **Fundus** → most distant area from blood supply.
 - **Hartmann's pouch** → friction with the obstructive agent.
 - Effects:
 - Peritonitis:
 - **After 48 hours** → localized peritonitis.
 - **Before 48 hours** → generalized peritonitis (very rare).
 - Fistula:
 - 1) Internal:
 - Sites:
 - ✓ CDB → Mirizzi's syndrome.
 - ✓ Transverse colon → cholecystocolic fistula.
 - ✓ Duodenum → cholecystoduodenal fistula → gall stone ileus.
 - 2) External: with skin (very rare).
5. Jaundice (due to affection of the CBD):
 - Ascending cholangiohepatitis.
 - A stone in Hartmann's pouch pressing upon CBD (Mirizzi syndrome)
 - An associated stone in the CBD.

6. Emphysematous cholecystitis:

- e.g. *Clostridium welchii* in old diabetic patient☑.
- Infection is highly virulent and leads to early gangrene of gall bladder.
- Diagnosed Infection by gas gangrene organism by X-Ray: gas inside the lumen☑

Gall stone ileus

(Seen most frequently in women > 70☑)

- **Cause:**
 - Stone in GB → fistula between GB and duodenum☑ → stone in small intestine → obstruction (at ileocecal valve or Meckel's) → start as mechanical obstruction → distention → ileus.
- **C/P:** see intestinal obstruction.
- **Plain X-Ray** may show (in addition to multiple fluid levels):
 - Gases in biliary tree (aerobilia)☑
 - Stone.
- **Treatment** (no conservative treatment):
 - Treatment of IO → resuscitation and monitoring + stone removal.
 - Treatment of GB as a mass.
 - Repair of fistula (to avoid recurrence).

DD*Of acute abdomen*

- 1) **Acute appendicitis** (with subhepatic appendix).
- 2) **Acute pancreatitis** → pain ↑ in crescendo up to neurogenic shock, cyano-icterus with ↑ serum amylase.
- 3) **Acutely perforated DU** → peptic dyspepsia.
- 4) **Right pyelonephritis.**
- 5) **Amoebic hepatitis.**

Investigations**A. For diagnosis**1. **Laboratory:**

- **CBC:** PMN leucocytosis.
- **LFTs:** usually normal.

2. **Radiological:**

- **U/S: (investigation of choice)**

➤ **Gall bladder:**

- Stones obstructing cystic duct with sensitivity reaching 98% Distension.
- Thickened wall.
- Serosal edema. – micro abscesses.

➤ **CBD:**

- Diameter of the CBD (normal 0.6 mm)
- Stones.

➤ **DD of radio-opacity in Rt. hypochondrium:**

1. Stone in the Rt. kidney.
2. Fecolith in intestine.
3. Calcified L.N.
4. Phlebolith.

- **HIDA:**

➤ **Scanning of the liver & gall bladder:**

- Is superior to U/S (**most accurate but not practical**☑).
- If the CBD is visualized while the GB is not seen, this is diagnostic for acute calculous cholecystitis.

**Acute cholecystitis**

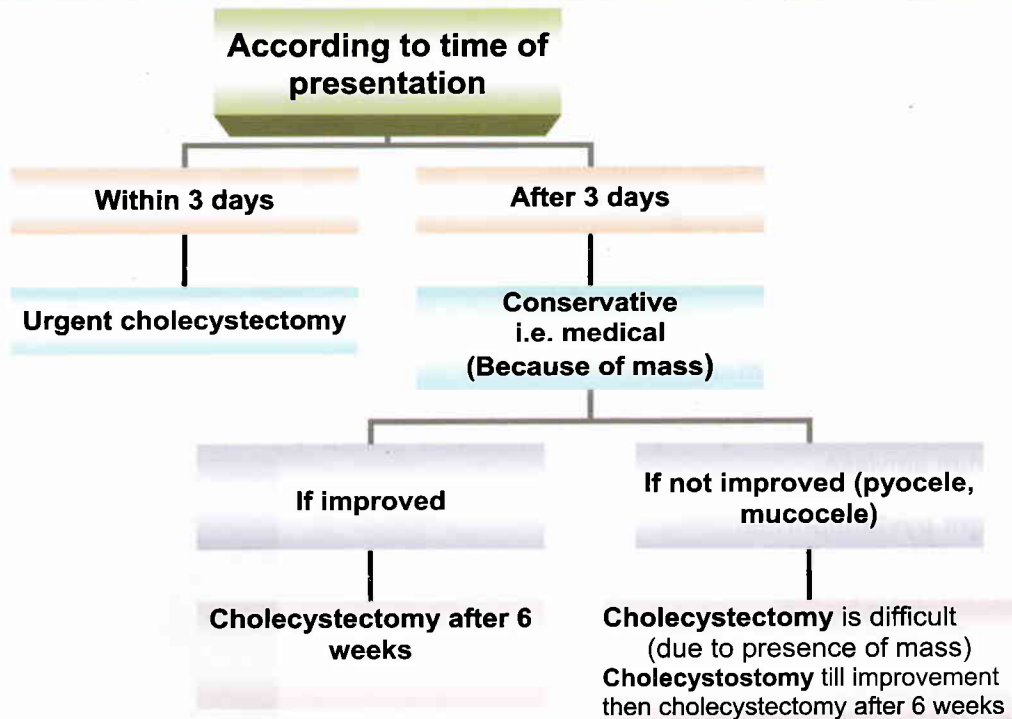
B. To exclude D D e.g:

- ECG for ischemic heart
- Serum amylase for acute pancreatitis

Treatment

1. Treatment of acute cholecystitis.
2. Treatment of complications.

Treatment of Acute Cholecystitis

**⇒ According to time of presentation:**

A- Within 1st 3 days: Cholecystectomy ☒.
15% of patients undergoing cholecystectomy have had CBD stones ☒.

▪ **Provided that:**

- 1- Sure diagnosis.
- 2- Good surgeon.
- 3- Fit patient.

▪ **Advantages:**

1. Early surgery is not difficult as adhesions are fibrinous and not fibrous.
2. Avoid possible future complications.
3. One day hospital admission with early return to work.

**B- After 48 hours: Medical treatment****1. Put the patient in modified Fowler's (semi-sitting) position as it:**

- i. Relaxes the abdominal wall.
- ii. Lowers the intra-abdominal pressure.
- iii. Drains pus into the pelvis in case of rupture of the GB.

2. Feeding: Stop oral feeding & IV fluids.**3. Ryle:** tube suction.**4. Hot application:** to the Rt. hypochondrium.

5. **Antispasmodics:** to relax the sphincter of Oddi.
6. **Sedatives:** e.g. Pethidine because morphia is contraindicated as it causes spasm of the sphincter of Oddi, or NSAIDs.
7. **Antibiotics:** (start once diagnosis is made)
 - i. Broad spectrum antibiotics are most appropriate e.g. gentamycin, cephazoline, Quinolones (high serum concentration).
 - ii. Antibiotics e.g. **3rd generation Cephalosporins** is contraindicated as its concentration in bile is high.
8. **Observation:** Pulse, temp, severity of pain & early manifestations of complications

- ⇒ **If improved** → cholecystectomy after 6 weeks.
- ⇒ **If not improved** → cholecystectomy and if difficult → cholecystostomy till improvement then cholecystectomy after 6 weeks.
- ⇒ **NB:** it is the trend now for treatment of acute cholecystitis by early surgery.

➤ **Those who prefer conservative treatment claim that:**

1. Most patients will settle on conservative treatment.
2. Surgery in acute stage is difficult as tissues are friable and edematous leading to increased liability for injury of the surrounding structures.

Treatment of the Complications

1. **Treatment of cholecystocolic fistula:**
 - Proximal colostomy.
 - Later on cholecystectomy & closure of the fistula.
2. **Treatment of Mirizzi Syndrome:**
 - ERCP, sphincterotomy, dormia basket.
 - Later on cholecystectomy.
3. **Gall stone ileus:**
 - Treatment of the intestinal obstruction:
Resuscitation & Monitoring + deal with the stone:
 - Crushing of the stone.
 - Milking to pass ileocaecal valve.
 - Removal by enterostomy.
 - Resection & anastomosis.
 - Later on cholecystectomy.
4. **Empyema:** Treated by urgent cholecystectomy.

B- Acute Non-calicular Cholecystitis

Etiology

- Commonly in immunocompromised patients.
- It is a rare condition which may occur in:
 - 1- Major burns or major trauma.
 - 2- Prolonged TPN.
 - 3- Infections as typhoid & brucella.
- The actual causes and pathogenesis are unclear.
- Change in the composition of bile or ischemia of gall bladder may be responsible.

- ▶ immunocompromised present in ICU
C/O FAHM+ Biliary colic
- ▶ TTT: Urgent cholecystectomy with poor prognosis

Clinical Picture

- Similar to acute calcular cholecystitis but the diagnosis is usually delayed & difficult because it is usually unsuspected, so it is a serious condition.

Investigations

- **U/S:** diagnostic.

Treatment

- **Urgent cholecystectomy**.

Prognosis

Mortality is higher than that of acute cholecystitis.

Chronic Cholecystitis

A- Chronic Calcular Cholecystitis

Etiology

- **Microorganisms:** E.Coli, Staph, Strept, Salmonella.
- **Route of infection:** direct or blood spread.
- **Predisposing factors:**
 1. Persistent predisposing factors as stones.
 2. Inadequate treatment of acute attack.
 3. Bad general condition.

- ▶ **Etiology:** E.coli + direct or blood spread
- ▶ **C/P:** 6F, biliary dyspepsia, biliary colic & +ve Murphy's sign
- ▶ **Comp.:** Porcelain GB (precancerous), GB stones, GB carcinoma
- ▶ **Invest.:** U/S, for comp., to exclude triads
- ▶ **TTT:** Cholecystectomy + TTT of complications

Pathology

Types

- Calcular 90%
- Non calcular 10%

Macroscopic Picture

- **Gall Bladder:**
 1. Shape: deformed.
 2. Lumen: may contain stones.
 3. Wall:
 - All layers: thickened by fibrosis, rarely calcified.
 - Serosa: loss of peritoneal luster.
- **Cystic LN of Lund:** enlarged.
- **Liver:** subcapsular fibrosis of the liver around the gall bladder bed.

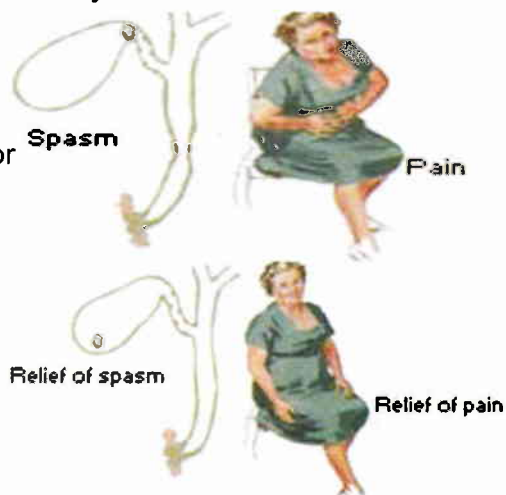
Microscopic Picture

- Fibrous tissue, end-arteritis obliterans.

Clinical Picture

Symptoms

1. **Fat (biliary) dyspepsia:**
Abdominal distention & excessive eructation after fatty meals.
 2. **Recurrent attacks of biliary pain:** (due to stone migration in the cystic duct)
 - Onset: sudden.
 - Site: Rt. hypochondrium.
 - Referred to: Rt. shoulder[□], the back & inferior angle of the Rt. Scapula (along intercostal, phrenic & suprascapular N = C₃, C₄)
 - Precipitated by: fatty meals.
 - Relieved by: antispasmodics.
 - Associated symptoms: nausea & vomiting in severe attacks.
- ⇒ If the attack persists: acute cholecystitis
3. **Reflex symptoms:** Retrosternal chest pain.



Signs

- **General:** (6F: Female, Forty or Fifty, Fatty, Filthy, Fertile)

- **Local:**

- **Murphy's sign[Ⓜ]:**

The GB area is palpated while the patient is asked to take deep breath → the patient will catch her breath.

- **Tenderness in the Rt. hypochondrium.**

It may be a part of Saint's triad or Wilkie's triad:

- **Saint triad[Ⓜ]:** (CC-HH-DD)

Chronic calcular cholecystitis, hiatus hernia, diverticular disease of the colon.

- **Wilkie's triad[Ⓜ]:**

Chronic cholecystitis, chronic PU, chronic appendicitis.

- Fat dyspepsia is common with reflux (GERD) than with chronic calcular cholecystitis

DD

From other causes of dyspepsia:

- Chronic appendicitis.
- Chronic peptic ulcer.
- Chronic pancreatitis.
- Cancer stomach.
- Hiatus hernia.
- Liver cirrhosis.
- Colonic dyspepsia.

Complications

➔ General

- **Chronic septic focus:** toxic myocarditis, arthritis, anemia.

➔ Local

1. **Acute exacerbation.**

2. **GB stones:** complications

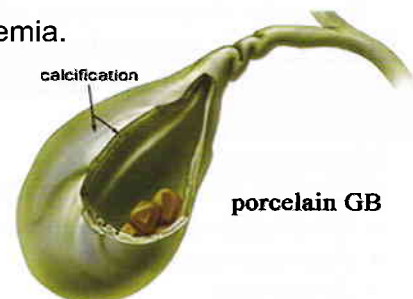
3. **Cardiac link:**

- Pseudo-anginal pain.
- Unknown etiology.
- Condition improves after cholecystectomy

4. **Porcelain GB[Ⓜ]:**

- It occurs as a result of low grade chronic inflammation.
- Gall stones are almost always present in cases of gall bladder calcification.
- It ↑ incidence of GB carcinoma.
- Prophylactic cholecystectomy is indicated.

5. **Carcinoma of the GB.**



Investigations

I. **For diagnosis:**

- **Abdominal U/S:**

- GB is shrunken & fibrotic + presence of stones
- Stone in CBD or dilated CBD

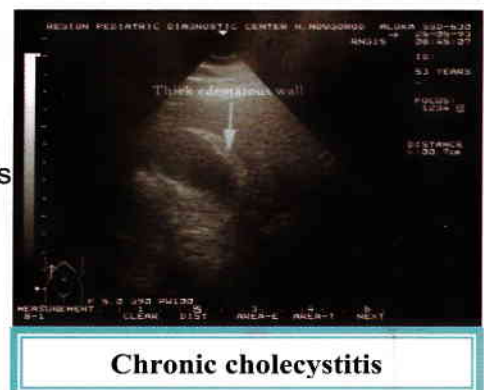
- **Dynamic U/S:**

- To detect if GB changes in size before & after meals
- To detect the function.

- **Oral Cholecystography:** not done now.

- **Plain X-Ray**

- Only 10-15% of stones are radiopaque.



2. For complications:

- **LFTs:**
 - (If high direct bilirubin & ALP → Stone in CBD → ERCP is needed)
- **ERCP:**
 - If patient is jaundiced or there is history of jaundice

3. To exclude wilkies or saint`s triad:

- **Endoscopy**
- **Barium meal**

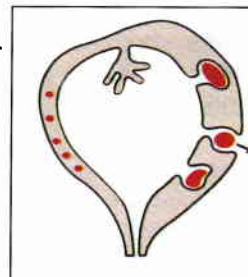
Treatment

- **Cholecystectomy** (NB: Modified Fowler's position is preferred during cholecystectomy).
- **Treatment of complications e.g. obstructive jaundice (see later).**

B- Chronic Non-calicular Cholecystitis

Types of Chronic Noncalicular Cholecystitis

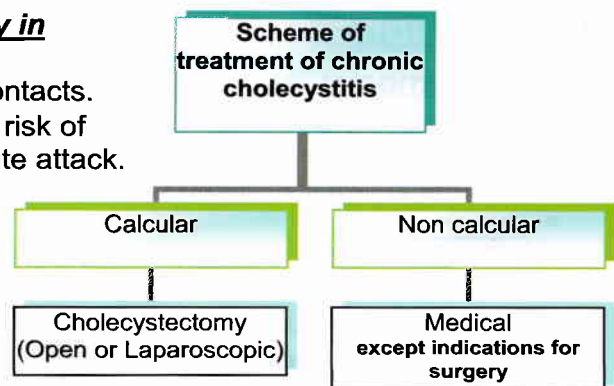
1. **Infection:**
 - e.g. typhoid carrier (salmonella present in GB).
2. **Cholesterosis:**
 - Deposition of cholesterol crystals in submucosa due to error in cholesterol metabolism → **Strawberry gall bladder**TM.
3. **Biliary dyskinesia:**
 - Sphincter does not relax when GB contracts.
4. **Cholecystitis glandularis proliferans**
5. **Diverticulosis of the gall bladder**



Types of cholecystitis
glandularis
proliferans
(polypus,
intramural or
diverticular stones
and fistula)

Treatment

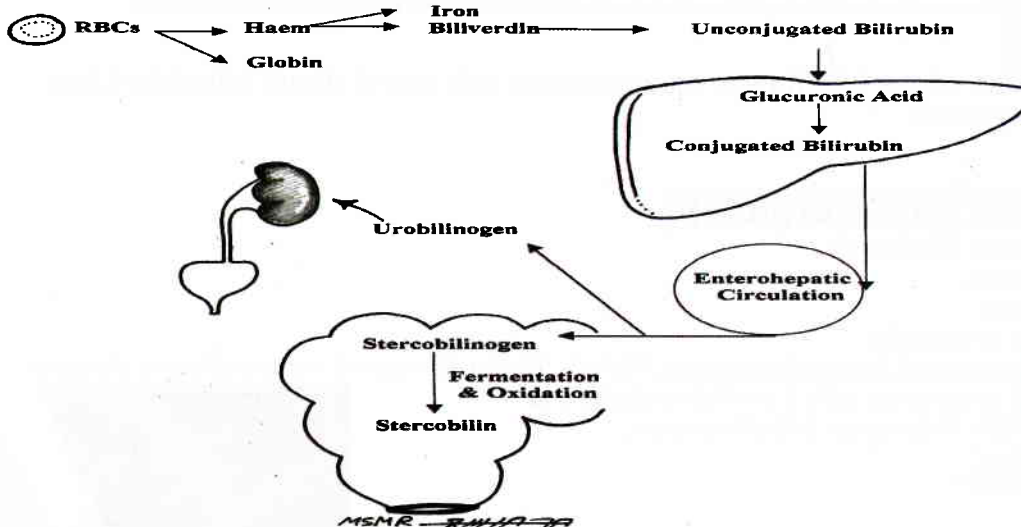
- **Mainly medical**
 - Control diet; avoid fatty meal and heavy bulky food.
 - Cholagogues as magnesium sulphate.
 - Antispasmodics.
 - Antibiotics.
- **Surgical (cholecystectomy) is indicated in:**
 - Failure of medical treatment after 6 months.
 - DM (chronic infection causes bad control of DM).
 - Typhoid carrier:
 - Detected by stool culture.
 - ➔ **Why do we do cholecystectomy in typhoid carrier?**
 - Seeking for benefit of the contacts.
 - The patient carries a higher risk of perforation especially in acute attack.
 - Non functioning GB.



Jaundice

Definition

- Yellowish discoloration of sclera, mm & body fluids (except the brain, CSF, tears, saliva & milk) due to excess bilirubin in the blood.
- Normal level of bilirubin 0.2 - 0.7 mg%.
- Hyperbilirubinemia > 2.5 mg%.



**Bilirubin
Metabolism**

Hemolytic Jaundice

Characters

- ↑ Serum bilirubin (mainly indirect). Bile salts do not accumulate in serum.
- ↓ Direct bilirubin in urine (acholuric jaundice).
- ↑ Urobilin in urine & ↑ stercobilin in stools.

Causes

- Hemolytic anemia.
- Erythroblastosis fetalis.
- RBCs destruction by:
 - Poison e.g. snake venom.
 - Infections e.g. malaria & septicemia.

Hepatocellular Jaundice

Pathophysiology

- Disease or toxin → liver cell damage → can not conjugate all indirect bilirubin → ↑ in the blood.
- Liver cells swell (VH) → biliary obstruction (↑ bile salts, ↑ direct bilirubin).

Characters

- ↑ Serum bilirubin (both types) → (Biphasic).
- Urine contains direct bilirubin & bile salts.
- ↓ Urobilin in urine & ↓ stercobilin in stools.
- Steatorrhea.

Causes

- Viral hepatitis.
- Liver cirrhosis.
- Toxins e.g. halothane, phenothiazines, testosterone derivatives, OCP's, oral antidiabetics, arsenicals.
- Infections e.g. septicemia, amoebic hepatitis & yellow fever.

Obstructive Jaundice

Definition

- **Obstruction of the bile flow** to the duodenum with rise of **direct bilirubin** & bile salts in the blood.

Etiology

Intra-hepatic (non-surgical)

- Viral hepatitis (Watson \$).
- Liver cirrhosis.
- Liver tumors.
- Sclerosing cholangitis.
- Familial conjugated hyperbilirubinemia: Rotor's, Dubin Johnson & veno-occlusive disease.
- Hormonal: pregnancy, pills & methyl testosterone.
- Drugs: PASA, Rifampicin & Erythromycin.

Extrahepatic

I. Lumen:

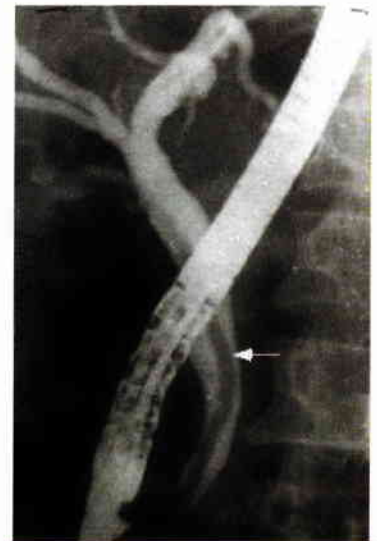
- **Calcular obstructive jaundice**: due to:
 1. Stone comes from the gall bladder. (15% of patients with calcular cholecystitis have stones in the CBD)
 2. Rarely primary in the CBD due to stricture.
- **Parasitic infestation**: as ascaris & fasciola.

2. Wall: (stricture of CBD due to)

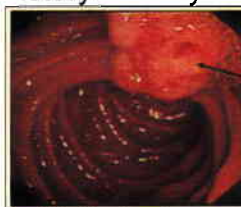
- **Congenital**:
 - Biliary atresia & choledochal cyst.
- **Acquired**:
 1. Inflammation → sclerosing cholangitis
 2. Trauma → post operative.
 3. Malignant stricture as cholangiocarcinoma.

3. Pressure from outside

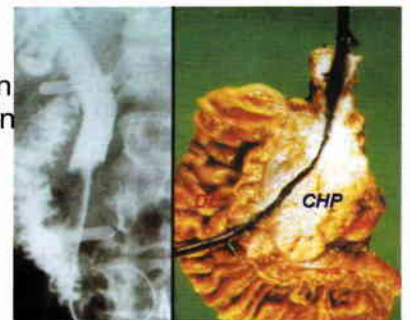
- **1ry tumors**:
 1. Cancer head of pancreas.
 2. Periapillary carcinoma:
- **Secondaries in LNs**: in porta hepatis, the obstruction tends to be short, segmental, concentric and occurs in patient with a clinical history of a 1ry neoplasm elsewhere.



Ascariasis worms causing obstructive jaundice



Endoscopic picture: ampullary carcinoma



Calcular Obstructive Jaundice (Cholelithiasis)

Pathology

Formation

1. In most cases stone in the CBD is originally formed in the gall bladder and has passed through the cystic duct into the common bile duct
2. Primary stones in the common bile duct are rare and occur when there is prolonged stasis and infection

Sequelae

- 1- No effect if the stone remains floating in CBD without obstruction
- 2- Passage to the duodenum. A small stone, usually less than 3mm, can pass spontaneously through the sphincter of Oddi. During its passage it may produce acute pancreatitis
- 3- Obstruction of CBD
 - Obstructive jaundice
 - Dilatation of the bile duct and intrahepatic biliary radicles.
 - In the presence of prolonged obstruction and high pressure in the bile ducts, bile secretion by the liver stops. The bile ducts become full of mucous (white bile).
 - **Complication:**
 - Bleeding tendency with failure of absorption of fat-soluble vitamins including vitamin K
 - Cholangitis due to infection by Gram-negative bacilli
 - Septicaemia and hepatorenal failure
 - Acute pancreatitis if the stone impacts in the lower part at the common channel of pancreatic duct and CBD
 - Biliary cirrhosis is uncommon in prolonged cases where obstruction is intermittent or incomplete

- **C/P:** - 6F, Slowly progressive jaundice, dark urine, clay colored stool, biliary colic, itching
- Charcot's triad, Reynold's pentad,
 - non-distended GB (80%), +ve Murphy's sign
- **Comp.:** OMUMI
- **Invest.:** -Lab.: LFTs(↑direct bil.), urine, stool
- Rad.: U/S, ERCP, PTC, MRCP
- **TTT:** Preoperative preparations then
- **ERCP:** Sphincterotomy + dormia basket
 - **Open:** if failed ERCP
- Cholecystectomy + Choledocholithotomy

Clinical Picture

Type of patient

- **6F** (Female, Fatty, Fertile, Flatulent, Forty or Fifty)

Symptoms

➤ It may be silent

1) Jaundice

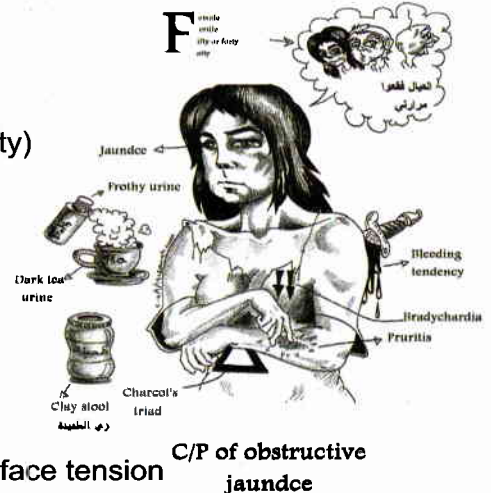
- Onset: slowly progressive
- Course: Intermittent, usually does not reach severe degree.
- Duration: variable

2) Urine

- Dark → excess direct bilirubin
- Frothy → excess bile salts → ↓ surface tension

3) Stool

- Bulky offensive clay colored stool.



4) Pain (biliary colic)

- Site: Rt. Hypochondrium, epigastrium
- Radiation: Rt. Shoulder, back
- Character: recurrent attacks
- What ↑: fatty meals
- What ↓: antispasmodics
- Associated symptoms: there may be nausea & vomiting.

5) Fever: intermittent with rigors (ascending cholangitis)

Charcot's triad[☐]: indicates ascending cholangitis :

- Pain.
- Jaundice.
- Fever and rigors.

Reynold's pentad[☐] : as above + altered mental state & shock

6) Itching: specially in limbs due to ↑ bile salts and PG in skin.**7) Bleeding tendency:** due to Malabsorption of Vit. K, improved by IV Vit. K.

- CBD has no smooth Ms. → so, it can be presented by dull aching pain.
- G.B. has smooth Ms. → so, it can be presented by colicky pain.

Signs**A. General**

- 4- 6F obese
- 5- Vital signs:
 - Temperature: high if associated with cholangitis.
 - Pulse: bradycardia (bile salts suppresses SAN & myocardium).
 - BP: hypotension (vasodilator effect of bile salts).
- 6- Jaundice: may not be severe.
- 7- Purpura and ecchymosis.

B. Local**1- GB:**

- Usually 80% not distended[☐] (chronic inflammation → fibrosis).
- Murphy's sign is +ve.

Courvoisier's law[☐]:

- In obstructive jaundice if gall bladder is palpable it is most probably non-calicular with exceptions
 - a. Metabolic stone (in healthy GB) → Distended non tender GB.
 - b. Associated stone in cystic duct
 - c. Big stone in Hartmann's pouch obstructing CBD from outside and mucocoele of G.B.

2- Liver: enlarged non tender due to hydrohepatosis
i.e. (retention of bile in liver)**3- Spleen:** normally not felt, except in hemolytic stones**Complications**

- See before

Investigations**Laboratory**▪ **LFTs:**

- **Bilirubin:** ↑ serum bilirubin mainly direct fraction[☐]. It usually does not exceed 10mg/dl and its level may fluctuate.
- **Alkaline phosphatase [normally (3-13) king Armstrong]:** > 30 KA units
- **SGOT & SGPT:** No rise unless cholangiohepatitis occurs.

- γ **GT and 5-nucleotidase**: Both are elevated
- **PT**: Prolonged, improved by IV vitamin K
- **Stool**:
 - Clay colored, bulky offensive.
- **Urine**:
 - Dark colored.
 - Frothy.
 - Bilirubin: $\uparrow$ direct $> 10 \text{ mg\%}$.
- **BUN & creatinine**: $\uparrow$ in hepatorenal failure.
- **CBC**: to exclude hemolytic anemia, increase TLC if there is cholangitis (polymorph-nuclear leucocytosis).

Radiological

- **Abdominal U/S**: (*U/S is less sensitive to lower part of CBD & the first to be done*)
 - CBD stones are suggested by CBD diameter $> 8 \text{ cm}$ on U/S.
 - Dilated intrahepatic biliary radicals.
 - Chronically inflamed GB with stones.
- **ERCP**: (*indicated in OJ with suspected lesion in lower end of the CBD*)
 - Both common bile ducts and pancreatic duct will be visualized.
 - Stone will appear as a filling defect.

ERCP shouldn't be done during an acute attack of cholangitis unless a therapeutic procedure to drain bile is planned because the rise of pressure during injection of the contrast material may lead to severe systemic sepsis.

- **PTC**: (*indicated in obstructive jaundice with suspected lesion in upper end of CBD*)
 - Visualize extra and intrahepatic biliary system.
 - **Indications**:
 - a. Impacted stone.
 - b. Arrest of dye due to stricture.
 - c. Failure of cannulation of duodenal papilla).
- **MRCP**: (good diagnostic value but not therapeutic)

DD

- **From other causes of obstructive jaundice**: $\rightarrow$ malignant obstructive jaundice.
- **From other causes of jaundice**:
 1. Hemolytic jaundice (lemon yellow jaundice - dark stool - $\uparrow$ indirect bilirubin)
 2. Hepatocellular jaundice (Biphasic $\uparrow$).
- **Yellow discoloration of sclera**:
 1. Dark races.
 2. Carotinemia.
 3. Atebrine intake.

Treatment (It is a surgical emergency)

Preoperative Preparations

1. **Correct clotting dysfunction**:
 - I.V vitamin K & patient should be checked before operation.
 - Fresh blood transfusion.
2. **Guard against liver cell failure**:
 - High intake of glucose
 - Broad spectrum antibiotics (e.g. cephalosporins) if there is evidence of cholangitis
3. **Guard against renal failure**:
 - Adequate hydration by IV fluids.
 - IV mannitol.
 - Oral bile salts.

Definitive Treatment

1. Aim:

- To relieve biliary obstruction by removal of CBD stones (1st priority)
- To remove the GB, that is usually the source of CBD stones (2nd priority).

2. Methods:

Methods

➤ If technical support permits:

→ ERCP:

- Sphincterotomy by diathermy (at 11 o'clock to avoid injury to the blood supply).
- Removal of stone(s) by dormia basket.
- Later on, cholecystectomy
- A large stone can be fragmented before removal either by mechanical, electrohydraulic or laser lithotripsy

- If failed ERCP we do PTC → balloon dilatation then extraction

Complications of ERCP:

1. Bleeding in 2-9% may be due to coagulation disorder.
2. Acute cholangitis in 1-3%.
3. Pancreatitis in 1-4%.

➤ If failed ERCP or not available:

- Cholecystectomy + choledocholithotomy (operative exploration of CBD and removal of the stones)

NB.

- Additional procedures to prevent future obstruction:

▪ Indications:

- Impacted stone in the lower end of CBD
- Stricture of CBD.
- Inaccessible stones.

or

▪ Procedure:

- If CBD > 2 cm → choledochoduodenostomy (end-to-side) or (side-to-side)
- If CBD < 2 cm → transduodenal (not done now) sphincteroplasty or choledochojejunostomy.

Malignant Obstructive Jaundice

Clinical Picture

Type of patient

- Usually elderly male

Symptoms

- It may be silent

1) Jaundice

- Onset: gradual
- Course: rapidly progressive in cancer head of pancreas & intermittent in periampullary carcinoma (due to sloughing of the tumor).
- Duration: short.

2) Urine

- Dark → excess direct bilirubin
- Frothy → excess bile salts

3) Stool

- Bulky offensive clay colored stool.
- Silver stool in periampullary carcinoma.

4) Pain (biliary colic)

- **Pain is late**
- Site: epigastrium
- Radiation: back
- Character: boring
- What ↑: lying down
- What ↓: leaning forward

5) Fever: afebrile unless cholangitis develops

(Charcot's triad, Reynold's pentad → see calculous obstructive jaundice)

6) Itching.

7) Marked weight loss due to pancreatic cachexia

8) Bleeding tendency.

► C/P: as calculous OJ but

Old ♂ with RAPIDLY progressive jaundice, cachexia, distended GB, late pain

► Invest.: as above + CT, tumor markers, Ba meal

► TTT: • Preoperative preparations then:

a- **Unfit for surgery** → Stent

b- **Fit for surgery** → Whipple's operation

Signs

A-General

2. Elderly male with cachexia.
3. Vital signs:
 - Temperature: usually normal except if cholangitis develops.
 - Pulse: bradycardia.
 - BP: hypotension.
3. Jaundice: may be severe.
4. Itching marks.
5. Purpura and ecchymosis.
6. LNs: enlarged supraclavicular LNs (Virchow's) in advanced malignancy.

B- Local

1- GB:

- Commonly distended & tender (healthy gall bladder).

► In case of obstructive jaundice if gall bladder is palpable, it is most probably non-calculous (malignant) with exceptions:

- a. Associated chronic cholecystitis
- b. Malignant obstruction above cystic duct (Klatskin's tumor)
- c. Previous cholecystectomy.

- 2- **Liver:** either:
 - Enlarged non tender due to hydrohepatosis.
 - Enlarged, tender, nodular & hard due to metastasis.
- 3- **Spleen:** enlarged if obstruction is due to lymphoma.
- 4- **Ascites:**
 - Due to infiltration of portal vein or liver metastasis (malignant ascites).
- 5- **DRE:**
 - $\pm$ Krukenburg.
 - $\pm$ peritoneal nodules.

Investigations

Laboratory

- **LFTs:**
 - **Bilirubin:** $\uparrow$ serum bilirubin specially direct fraction $\square$.
 - **Alkaline phosphatase:** > 30 KA units
 - **SGOT & SGPT:** No rise unless cholangiohepatitis occurs.
 - γ **GT and 5-nucleotidase:** Both are elevated
 - **PT:** Prolonged.
- **Stool:**
 - Clay colored, bulky & offensive.
 - Contains excess undigested fat.
 - No or $\downarrow$ stercobilinogen $\square$.
 - $\pm$ Occult blood in periamupullary carcinoma.
- **Urine:**
 - Dark colored.
 - Frothy.
 - No or $\downarrow$ urobilinogen $\square$.
 - Bilirubin: $\uparrow$ direct > 10 mg% $\square$.
- **BUN & creatinine:** $\uparrow$ in hepatorenal failure.
- **CBC**

Radiological

- **Abdominal U/S:**
 - Dilated intrahepatic biliary radicals.
 - Dilated CBD.
 - A mass of head of the pancreas may be detected.
- **CT scan (indicated in suspected malignancy)**
 - Mass in the pancreas.
 - Liver deposits.
 - Metastatic LNs.
- **ERCP:**
 - Both common bile ducts and pancreatic duct will be visualized.
 - Detects any lesion of the ampulla and biopsy may be taken.
- **PTC:**
 - Visualize extra and intrahepatic biliary system
- **MRCP**

Special Investigations

A. **Tumor markers:**

- CEA, POFA, PCAA
- Prognostic value

B. **Barium meal:**

- Widening of the C-curve of the duodenum $\square$ (Cancer head of the pancreas)
- Inverted 3-shaped $\square$ (Peri-ampullary carcinoma).

DD

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- **From other causes of jaundice:**
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- **Yellow discoloration of sclera:**
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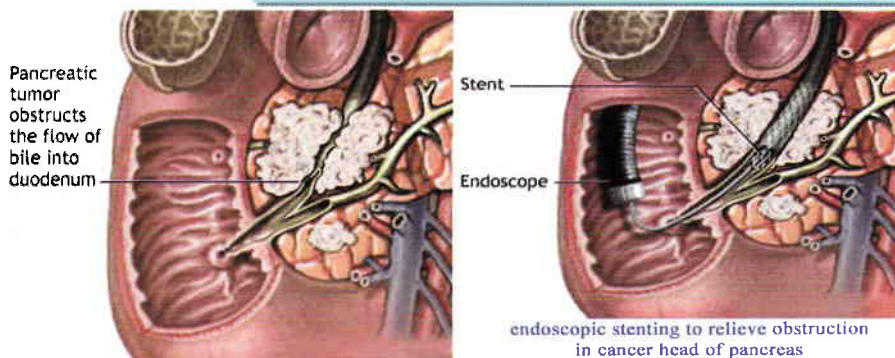
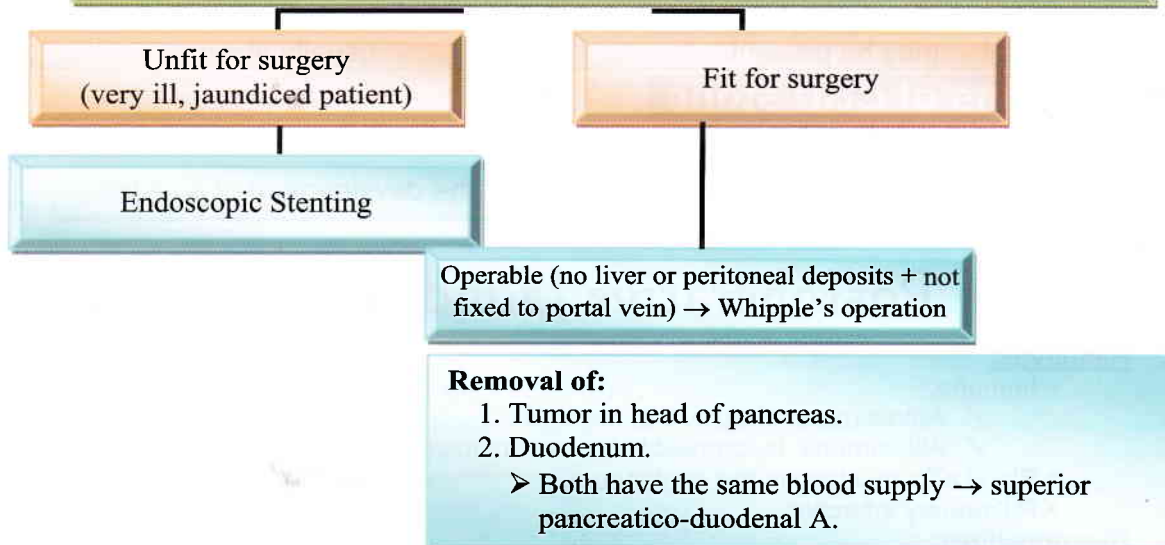
Treatment

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 - High intake of glucose
 - Broad spectrum antibiotics.
3. **Guard against renal failure:**
 - Adequate hydration by IV fluids.
 - IV mannitol.
 - Oral bile salts.

Operations

According to the general condition of the patient



	Calcular OJ	Malignant OJ
1. Type of patient	6 F	Elderly male
2. Jaundice	Slowly progressive, not severe intermittent,	Gradual, rapidly progressive or intermittent
3. Stool	Clay colored	Clay colored or silvery stool
4. Urine	▪ Dark due to excess direct bilirubin. ▪ Frothy due to excess bile salts → ↓ surface tension.	
5. Pain	▪ Recurrent attacks of dull aching pain ▪ Rt. Hypochondrium → Rt. Shoulder	▪ May be absent ▪ Boring ▪ Epigastrium → Back
What ↑ What ↓	Fatty meals Antispasmodics	Lying down Leaning forward
6. Weight loss	-ve	Marked
7. LNs	-ve	+ve (Virchow's LNs)
8. Liver	Enlarged, not tender	Enlarged, not tender or enlarged, nodular & tender
9. Spleen, ascites, P/R	-ve	+ve
10. U/S	Usually fibrosed gall bladder with stones	- distended gall bladder with thin wall
11. CT	- Gall stones	- Head neoplasm
12. ERCP	- Stone in CBD	- Irregular filling defect
13. Gall bladder	- Usually impalpable	- Commonly palpable
14. Fever	- may be present	- usually absent

Complications of CBD stone

- See complications of gall stones.

There is no relation between the size of the stone and the development of jaundice.

Postoperative Jaundice

A. Hemolytic:

- Immune:
 - ✓ Autoimmune (drugs): e.g. penicillin.
 - ✓ Alloimmune: Incompatible blood transfusion.
- Blood accumulating in the peritoneum.
- Pulmonary infarction.

B. Hepatocellular:

- Liver damage:
 - ✓ Operative stress upon liver disease.
 - ✓ Liver hypoxia during operation.
- Toxic: Anesthetic toxicity as fluthane.
- Infections:
 - ✓ Viral hepatitis.
 - ✓ Septicemia.

C. Obstructive:

- Leakage of bile into the peritoneal cavity.
- Stricture of the CBD.

Carcinoma of the Gall bladder

Incidence

- Uncommon neoplasm.
- More common in elderly females.

Predisposing Factors

- Gall stones in 90% of cases.

Pathology

- Adenocarcinoma (most common).
- Squamous cell carcinoma.
- Mixture of both.

Complications

Spread:

1. Direct:

- Liver and biliary passages.

2. Blood:

- Mainly → liver via portal circulation
- Rarely → lungs, bones, brain via systemic circulation.

3. Lymphatic:

- Early → Cystic LN of Lund and porta hepatis LNs
- Late → to Virchow's LNs (Troisier's sign).

4. Transcoelomic: either by

- Seeding or retrograde peritoneal lymphatic spread.

Clinical Picture

Pathological group

- Usually diagnosed after pathological examination of removed GB because of chronic calculous cholecystitis.

Obstructive group

- Acute cholecystitis: due to obstruction of cystic duct.
- Obstructive jaundice: due to obstruction of CBD either by tumor or metastatic LNs.

Mass group

- Mass in the Rt. hypochondrium.

Investigations

1. For diagnosis:

- CT and U/S scans:

May demonstrate the extent of disease, but more often they show only gall stones.

- U/S guided biopsy.

2. For staging: CT, CXR, US

3. For preoperative preparation: KFTs, LFTs, CBC,

Treatment

- If suspected during surgery:

- Cholecystectomy.
- Wedge resection of an adjacent 3-5 cm of normal liver.
- Dissection of the lymph nodes overlying the bile duct.

- Inoperable cases:

- Relieve jaundice by internal stenting.



A ring of calcification in the expected location of the gallbladder



Carcinoma of the GB

Stricture of the Bile Ducts

1- Congenital Biliary Atresia

(See before)

2- Traumatic Stricture

Etiology

It may be due to: most commonly postoperative[☑]. Usually follows cholecystectomy or less commonly choledocholithotomy.

- Complete ligation of CHD or CBD.
- Narrowing of the ducts due to its inclusion in a ligature.
- Devitalization of the duct due to rough dissection.
- Ischemia of the ducts.

Clinical Picture

- Presents by either: biliary fistula or jaundice following cholecystectomy.

Investigations

- ERCP and PTC: will localize the site of the stricture.

Treatment

- The remaining stump of the common hepatic duct is anastomosed to Roux-en-Y loop of jejunum.

3- Inflammatory (Sclerosing Cholangitis)

Definition

- It is an autoimmune[☑] disease that presents usually by obstructive jaundice and if the condition is untreated it will lead eventually to liver cell failure.

Etiology

- Unknown.
- Associated with ulcerative colitis[☑].

Pathology

- The intra and extrahepatic ducts are involved by multiple strictures.

Clinical Picture

- Obstructive jaundice.
- Liver cell failure in untreated cases.

Treatment

- Anastomosis of a dilated segment of the bile duct to a loop of jejunum.
- In some patients there is not available dilated segment suitable for anastomosis, these patients are candidates for liver transplantation (ttt of choice[☑]).

4- Neoplastic Stricture (Cholangiocarcinoma)

Incidence

- Uncommon neoplasm
- More common in elderly males☑.

Predisposing Factors

- 1- Calcular disease.
- 2- Ulcerative colitis☑.
- 3- Primary sclerosing cholangitis☑.
- 4- Choledochal cyst.
- 5- Parasitic infestations by Clonorchis sinensis☑.

Types

- Intrahepatic
- Extrahepatic

Clinical Picture

Symptoms

- Obstructive jaundice (90% of patients).
- Pain, cholangitis and anorexia (less common).

Signs

- 1- **Hepatomegaly.**
- 2- **GB** may be:
 - **Distended**, if the tumour is below the insertion of the cystic duct into the CBD.
 - **Collapsed**, if the tumour is at porta hepatis (*klatskin's tumour*☑).

Investigations

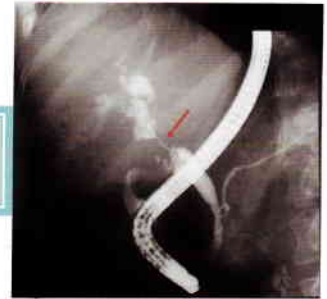
➤ For diagnosis:

- **U/S & CT scan:**
 - CT is the best investigation to detect biliary tumors.
 - Usually detect the dilated intrahepatic bile ducts
- **PTC:** bile samples may be obtained & may show malignant cells

Treatment

- Operable lesion at lower end of CBD**
→ treated by whipple's operation.
- Inoperable cases**
→ treated by cholecystojejunostomy

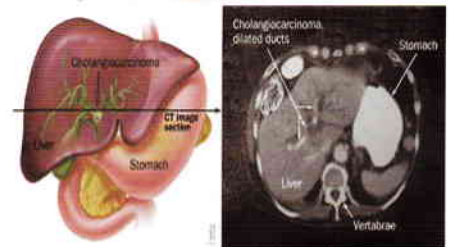
ERCP- bile duct carcinoma



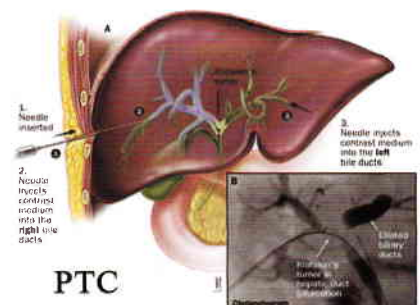
Benign stricture



Cholangiocarcinoma

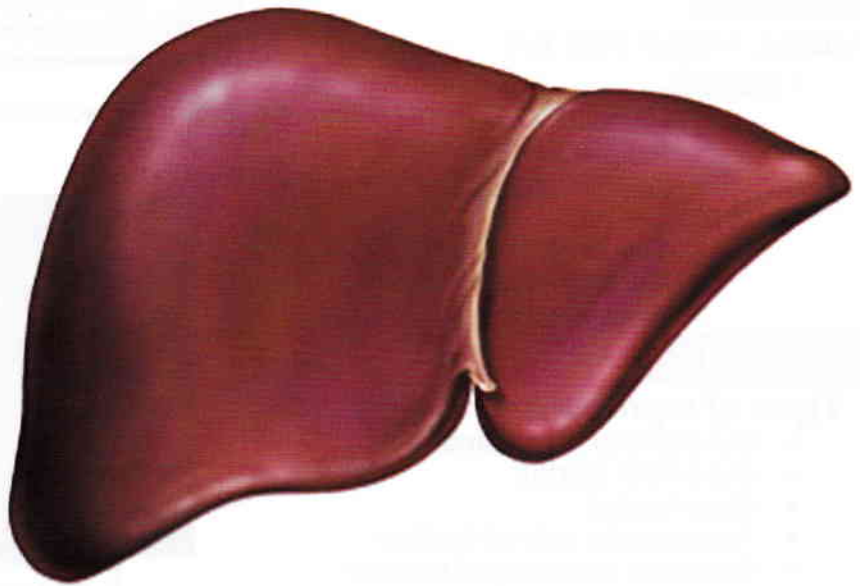


CT showing cholangiocarcinoma in the hilum of the liver



PTC

CHAPTER 8



THE LIVER

Liver Trauma

- The second common solid abdominal organ to be injured after spleen (both account for 75% of all blunt abdominal injuries)

Predisposing Factors

- **Liver enlargement:** which makes it more liable to trauma.
- **Diseases of the Liver:** which make it soft.

Etiology

Trauma, which may be:

- **Closed:**
 - Direct trauma: blunt trauma: e.g. motor car accident & falling from a height.
 - Indirect trauma: fracture ribs.
 - Spontaneous rupture: with pathological liver.
- **Open:**
 - Gun-shot wounds.
 - Puncture due to stabbing.
 - Iatrogenic: e.g. PTC or liver biopsy.

- ▶ 2nd common abdominal organ to be injured
- ▶ **Etiology:** Open, Closed, Iatrogenic
- ▶ **C/P:** History of trauma, hypovolemic shock, signs of blood in peritoneum (acute abdomen)
- ▶ **Invest.:** U/S & CT (main diagnostic tools), DPL
- ▶ **TTT:** 1- Management of polytrauma
2- Immediate laparotomy: stop bleeding (Pringle's manoeuvre) then deal acc. to injury

Pathology

Types of ruptured liver:

- Subcapsular hematoma.
- Superficial tear(s).
- Deep tear(s).
- Avulsion of a pole of the liver.
- Complete depulping of the liver.
- Injury of a vascular pedicle.
The most difficult injury is that of main hepatic veins because of difficult access.
- Hemobilia.



Multiple hepatic lacerations

Complications

1. **Hemorrhage:** (either internal or external)
 - Intraperitoneal hemorrhage can lead to shock.
 - Sub-capsular hematoma which may be infected leading to an abscess or hemobilia.
2. Infarction of liver tissues due to occlusion of liver blood supply by hematoma or abscess.
3. Biliary leakage leading to biliary peritonitis with subsequent abscess or cyst.
4. Associated abdominal or thoracic injuries.

Clinical Picture

History

- a. History of trauma to the upper abdomen or lower chest followed by abdominal pain.

General Examination (picture of hypovolemic shock)

- b. Rapid weak pulse, hypotension & subnormal temperature.
- c. Cold extremities & pallor.

We have to exclude other associated injuries of the chest, abdomen & head

Local Examination

1- Inspection:

- Bruises in the Rt. hypochondrium.
- Fracture of the Rt. lower ribs.
- Rigidity.

2- Palpation:

- Tenderness & guarding in the Rt. hypochondrium. Later becomes generalized.
- Rebound tenderness.

3- Percussion:

Shifting dullness.

4- Auscultation:

↓ intestinal sounds.

5- DRE:

Fullness in the rectovesical pouch & Douglas pouch.

The injury may be discovered during systematic exploration of the abdomen.

Investigations

I. Radiological:

▪ The most important diagnostic tools are:
- Abdominal U/S & CT.

▪ U/S and CT scan:

- Diagnostic → free blood in peritoneum & hematoma on the ruptured liver.
- Show pathological types and injuries to other organs.

▪ Plain X- ray:

- Fracture ribs.
- Elevated Rt. copula of the diaphragm.
- Obliterated psoas shadow.
- Multiple fluid levels.
- Giant fluid level due to peritoneal collections.



2. Instrumental:

- Selective hepatic angiography : may be helpful.
- Diagnostic peritoneal lavage (DPL).

3. Laboratory investigations:

- KFTs, LFTS, FBS, electrolytes, CBC.

Treatment

► Management of polytraumatized patient (advanced trauma life support)

1. Prehospital management ABCD

2. Hospital management

- a- Primary survey: ABCD
- b- Secondary survey:
 - Head to toe examination.
 - Resuscitation & monitoring.
 - History.
 - Investigations

3. Preoperative Preparation

- Blood transfusion and morphia.

4. Immediate Laparotomy

- Adequate exposure of the abdomen by longitudinal incision that can be extended in the chest.
- Systematic exploration of the abdomen.

The priority is to arrest bleeding:

- If the bleeding stops by the time of exploration, just put a drain
- We control the liver hemorrhage by a combination of temporary packing of the bleeding area and application of the Pringle's manoeuvre which is application of a vascular clamp to the free border of lesser omentum or holding it between 2 fingers (to occlude the hepatic artery and portal vein) for 20 minutes (failed Pringle's manoeuvre is due to hepatic vein bleeding) + fresh frozen plasma.
- Dealing with different types of injury
 - Suturing liver tears should be avoided (whenever possible) as it may cause a hematoma → infection or hemobilia.
 - However: in case of deep tears, we can not stop bleeding without suturing; in this case deeply placed mattress sutures supported by a pad of omentum is recommended.
 - If there is hematoma: we ligate damaged vessels and ducts and excise dead tissues.
 - Firm packing of difficult and inaccessible areas eg: with hepatic veins it may be the only method for temporary arrest of bleeding
- Multiple intraperitoneal drains: to avoid collections of blood and bile.
- Prophylactic antibiotics.

Conservative treatment may be used in haemodynamically stable patients. Follow them up by close observation of vital data, Hb \4 hours and daily U/S.

Prognosis

- Mortality rate of liver injury = 15 – 20 %. Rise to 70% if three organs are injured.
- Some blunt traumas can be treated conservatively, e.g. if CT shows minor hematomas and small tears. However there are indications to explore the patient such as evidence of ongoing blood loss, signs of generalized peritonitis.
- Penetrating injuries should be explored.

Liver Infections

1 - Hydatid Disease of the Liver

Geographical distribution

- Common in sheep rearing districts of Australia and South America, Greece and Turkey.
- In Middle East it is commonly seen in Iraq, Yemen and Libya.

Causes

- It is infection by larval stage of **Echinococcus granulosus**.

Life Cycle

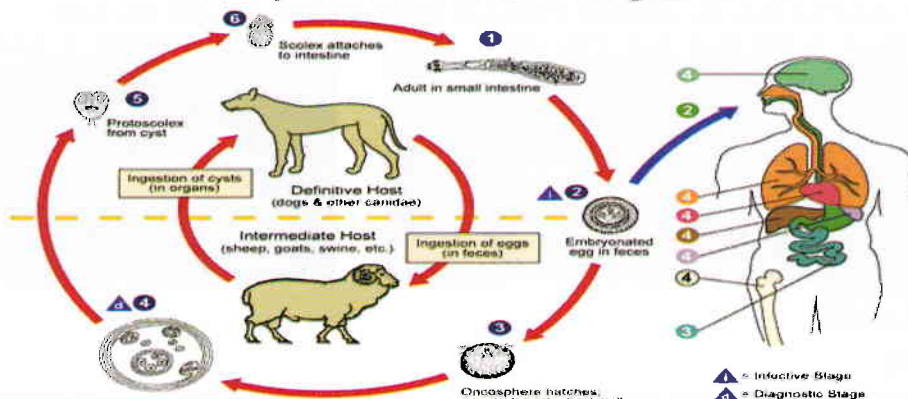
- The worm is composed of four segments with four terminal suckers.
- It lives in small intestine of the cats & dogs (**definitive host**).
- Eggs will pass with excreta → grass, vegetables in the soil.
- Eggs are ingested by sheep → hatch with release of larva (oncosphere).
- Larva will penetrate the wall of jejunum → Hydatid cyst.
- Dogs will eat the liver of the affected animals.

- **Causative org.:** Echinococcus granulosus
- Man is an accidental intermediate host
- **Path.:** Rt. Lobe, commonly solitary, 3 layers
- **C/P:** Asymptomatic {the most common} pain, swelling, comp.
- **Comp.:** as any cyst + spread
- **Invest.:** serology, imaging (U/S, CT, X-ray)
- **TTT:** **Medical** TTT + **surgery** {Enucleation + Omentoplasty} + **medical** TTT



Echinococcus granulosus (Tapeworm)

Life cycle of Echinococcus granulosus



Human Affection

- Man will be an **accidental intermediate host** (blind host).
- Man will be infected by:
 - Ingestion of eggs with dirty vegetables
 - Playing with dogs.
- Ova are digested in the duodenum → embryo travels through portal blood stream to reach the liver.
- The larva may escape the liver through the hepatic veins & reach systemic organs as lung, spleen & brain.

Pathology of Hydatid Cyst

Site

- Common in the Rt. lobe.

Number

- Common to be solitary or may be multiple

Layers of the Cyst (Double layer cyst wall)

1- Adventitia (pericyst)

- Grey in colour
- Fibrous layer formed by liver
- Adherent to liver

2- Laminated membrane (ectocyst)

- White in colour
- Formed by the parasite from germinal layer
- Separated from adventitia by plane of cleavage, but this plane is lost in liver infections

3- Germinal layer (endo-cyst):

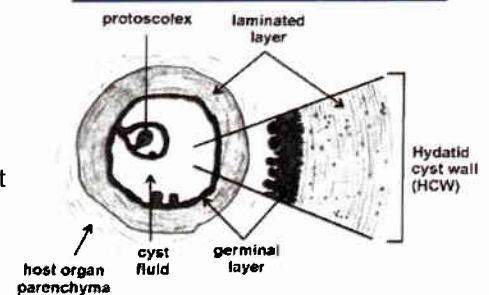
- It is the innermost layer & the only living part.
- It gives:
 - Scolices (head of future worms).
 - Brood capsule → Collection of scolices.
 - Daughter cyst:
 - May be formed outside the cyst or inside it.
 - Secrete hydatid fluid

NB:

It may occur after trauma & degeneration of the laminated layer

4- Hydatid fluid: it is crystal clear.

- Colourless and clear but may be yellowish if the cyst communicates with the bile duct
- Scolices are found in the fluid and may cause severe anaphylactic reaction if reaches circulation.



Clinical Picture

Type of Patient

- Common in people dealing with sheep.
- Common in certain countries as Saudi Arabia, Iraq, Libya & Sudan.

Symptoms

- Asymptomatic** (the most common).
- Chronic pain in Rt. upper quadrant.
- Swelling in the** upper abdomen.
- Picture of the **complications** (acute abdomen after minor trauma due to rupture of cyst into peritoneal cavity).
- May be other organ affection as in malignant hydatid → lung, brain ...

Signs

1. Swelling has the criteria of liver swelling.
2. Hydatid thrill might be felt on percussion (Due to vibration of daughter cysts in fluid) in 70% of cases
3. Hepatomegaly.

Fate & Complications

- The majority of cases, the cyst **gradually enlarges**.
- In others, the **parasite dies** and the **cyst is calcified**.
- Rarely, the cyst undergoes one of the following complications:
 1. **Rupture in**☐:
 - *Biliary tree* → obstructive jaundice (*the commonest*☐ 12-18%).
 - *Peritoneum* → severe anaphylaxis, urticaria (generalized hydatidosis) and diffuse peritonitis.
 - *Hepatic veins* → systemic affection especially lung.
 2. **Spread to other organs:** (lung, brain & bone)
 - More in *E. multilocularis*.
 3. **Secondary infection** (10-15%) Pyogenic liver abscess → painful.
 4. **Pressure:** On the surrounding structures.
 5. **Calcification**☐.
 6. **Hemorrhage**.

Investigations

A. For diagnosis:

1. Laboratory:

- **Serological:**
 1. Complement fixation test → + ve in 95% of cases.
 2. Haemagglutination test → + ve in 95% of cases.
 3. Casoni intra-dermal test☐ → + ve in 70% of cases (Not widely used now due to its low sensitivity and specificity).
 4. Immunoelectrophoresis is the most common serological test☐.
- **CBC:** Eosinophilia.
- **LFTs:** ↑ direct bilirubin & alkaline phosphatase (if jaundiced).

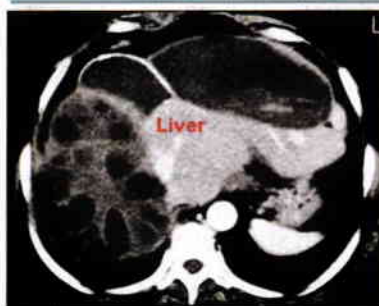
2. Radiological:

- **U/S:** can detect size site and number of cysts.
- **CT scan:** in endemic areas a liver cyst is considered hydatid cyst
- **Plain X-ray chest & abdomen:**
 - Elevated Rt. copula of the diaphragm.
 - Rt. Sided pleural effusion (± lung affection)
 - Calcified cyst.

Biopsy or aspiration are contraindicated.☐



Calcified hydatid cyst



B. For complications:

▪ ERCP:

- Indication: if the cyst ruptures into the bile duct causing obstructive jaundice
- Dye in the cyst → communication with the biliary tree.

Treatment

Is indicated to prevent progressive enlargement and rupture of

It is wise to give hydrocortisone before injection for a day before, during & after any surgical procedure

Mainly Surgical

Medical TTT + Surgery + Medical TTT

➔ **Is indicated to prevent progressive enlargement and rupture of cyst**

A. Sterilization of cyst:

1. Partial evacuation (as cyst is tense) by:

1. Double-way syringe.
2. Hydatid cone.

2. Injection of colloidal material in the cyst:

1. To kill the scolices.
2. Avoid anaphylaxis.

➤ Material used (left in the cyst for 15 minutes):

- a- Hypertonic saline 25%.
- b- Povidone iodine (betadine).

⇒ This is repeated till aspirate becomes clear.

⇒ Formalin is not used now as it causes fibrosis of the bile duct.

⇒ The cyst is then filled with isotonic sodium chloride solution.

B. Removal of cyst:

1. Enucleation (best treatment):

Enucleation + Omentoplasty

- Open the adventitia.
- Enucleate the cyst through the plane of separation between the adventitia & laminated membrane.

The cyst is surrounded by dark green towels to allow visualization of the scolices

C. Obliteration of the cavity:

- Omentoplasty (by pedicle omentum) to:

1. Obliterate the cavity.
2. Seal small biliary communications.

Medical Treatment

1. Indications:

1. Disseminated disease.
2. Preoperatively to avoid complications during surgery (4 days before operation).
3. Postoperatively to kill any spilled scolices and prevents recurrence for 1-3 months.
4. Small asymptomatic cyst.
5. Elderly patients.

2. Types:

I- **Mebendazole Or Albendazole** 400-600mg three times daily for 1 month.

- For large cysts albendazole should be prescribed. If there is no change in the cyst after a 6week therapeutic trial, then surgical management is appropriate.

Recently the use of Albendazol is better than Mebendazol as it is less toxic.

II- Hydrocortisone: by injection for a day before, during and after any surgical procedure.

3. Complications

1. Hepatotoxicity.
2. Bone marrow suppression.

Treatment of Complications

- a- Obstructive jaundice → explores CBD surgically or by endoscopic clearance of daughter cysts.
- b- Infected cyst (abscess) → drain & antibiotics.
- c- Calcified cyst → it can be followed on U/S, where active cyst becomes larger and more superficial).

2- Amoebic Liver Abscess

(Tropical abscess or dysenteric abscess)

Incidence

- It is more common than pyogenic liver abscess in endemic areas

Causative Organism

- **Entamoeba histolytica**
(As a complication of amoebic colitis).

Mode of Infection

- ▶ **Cysts are the infective form**
 1. **Ingestion** of food or drinks contaminated by **Entamoeba cyst**.
 2. Cyst hatches in intestine → trophozoite form releasing proteolytic enzymes.

Relation to Dysentery

- At any time after the onset of dysentery from few days to years.
- The more severe the attack of dysentery, the less incidence of abscess.

Pathology

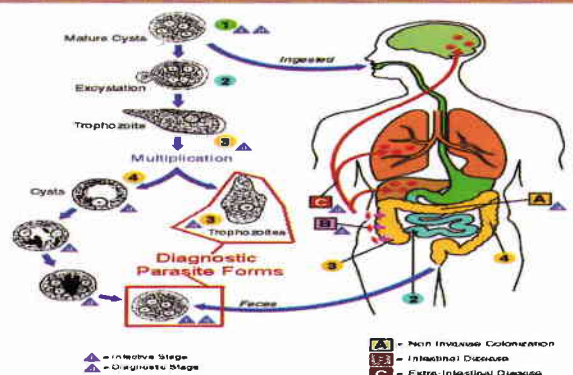
Site

- Rt. Lobe is affected more than Lt. lobe ✓:
 1. As it receives more portal blood.
 2. As *E. histolytica* is present mainly in caecum & Rt. colon → superior Mesenteric v. → Rt. branch & portal vein by streaming effect.
- The posterosuperior segment of the Rt. lobe of liver is the commonest site as the segmental branch of Rt. portal vein (to this segment) is in direct continuity with Rt. portal vein.

Number

- Solitary in 70% of cases.

- ▶ **Etiology:** Ingestion of food or drinks contaminated by *E. histolytica* cyst
- ▶ **Path.:** Rt. Lobe, solitary, anchovy sauce
- ▶ **Comp.:** as any cyst + chronicity
- ▶ **C/P:** young ♂+ as acute cholecystitis but with major pain and minimal fever with cough of anchovy like material
- ▶ **Invest.:** U/S, Metronidazole test, serology
- ▶ **TTT:** **A- Medical:** Metronidazole 800 mg 3 times daily for 7 – 10 days.
B- Surgical: aspiration, drainage



life cycle of *E. histolytica*
& its effect on the human body

Pus

- Usually sterile with brown chocolate colour and sometimes it is pinkish (anchovy sauce).
- The wall is shaggy and harbors amoeba.

Pathogenesis

Amoebic Hepatitis

- It is liquefactive necrosis of liver tissue.

➤ Fate:

- Healing by medical treatment. OR
- Coalescence together to form an abscess due to proteolytic enzymes and toxins production.

Amoebic Liver Abscess

- It is **not a true abscess** as there is no pyogenic membrane.

➤ Fate:

- **Complete Resolution:** is the rule.
- **Chronicity:** the abscess becomes surrounded by thick fibrous layer.
- **Complications:**

1. Rupture towards:

- Peritoneum → **peritonitis**.
- Sub-phrenic space → **Pleura** → Lung → coughing of chocolate sputum (commonest).

(When it ruptures upwards, it usually leads to lung abscess rather than empyema as the 2 layers of pleura are usually obliterated)

- Pericardium (amebic pericarditis & myocarditis).
- Stomach, duodenum or colon.
- Point on skin → **amoebiasis cutis**.

2. Secondary infection: forming pyogenic abscess.
3. Pressure effect: devitalizing adjacent liver cells.
4. Calcification.

Clinical Picture

Symptoms

Patient in endemic areas presents to us with picture similar to acute cholecystitis but with major pain and minimal fever

The most common presentation is with an attack of dysentery however history of an attack of dysentery may not obtained

- *There is predominance in young males.*
- **General:**
 1. Severe malaise, anaemia, loss of weight and earthy complexion.
 2. **Fever up to 38°C or more at night, profuse sweating & rigors** (especially with 2ry infection).
 3. Nausea & vomiting.
 4. Cough of **anchovy like material**.
- **Local: Pain**
 1. Severe, in the Rt. hypochondrium.
 2. Radiates to the Rt. Shoulder.
 3. ↑ by cough, stooping.
 4. ↓ by leaning on left side.

Signs

▪ General:

- 1- Low grade fever (*if high* → 2^{ry} infection → *Pyogenic abscess*).
- 2- Pallor & toxic look. (earthy look)
- 3- ± Jaundice (**severe cases**).
- 4- Rt. sided pleural effusion.

▪ Local:

1. **Inspection:** Rigidity & pitting edema over the lower right ribs.
2. **Palpation:** Tender hepatomegaly.
3. **Percussion:** Inter-costal tenderness, basal lung signs and dullness to percussion on the corresponding side.

Hepatocellular carcinoma in black Africans takes a rapidly progressive course that resembles amoebic hepatitis

DD

- Pyogenic liver abscess → high grade fever.

Investigations

1. Laboratory:

▪ CBC:

- PMN leucocytosis and anemia.

- **Serological:** CFT, IHA. Gel diffusion precipitative test is +ve in 95% of amoebic abscesses.

▪ **Stools analysis:**

- Useful in non-endemic areas.
- *Entamoeba histolytica* will be found.

▪ **Therapeutic test:**

- Toxic symptoms will improve on **metronidazole** as a therapeutic agent (diagnostic & therapeutic).

- Although diagnosis is by isolation of the parasite, patients with clinical signs of amoebic abscess will be treated empirically with metronidazole and investigated only if they do not respond.

▪ The most important diagnostic tools are:

- 1- U/S.
- 2- Metronidazole test.
- 3- Isolation of the parasite from the liver or stool.
- 4- Stool analysis in non-endemic areas.

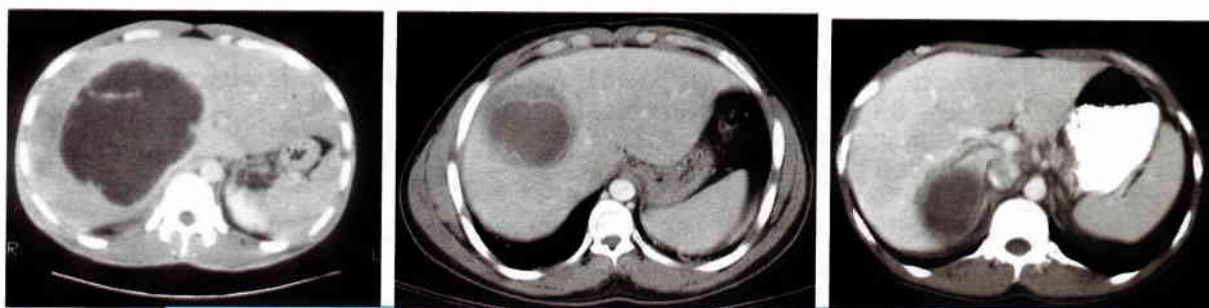
2. Radiological:

- **U/S & CT:** Detects number, site & size of abscess.

▪ **Plain X-ray:**

- Elevation of the right cupula of the diaphragm.
- Rt. sided pleural effusion.

In U/S and CT Amoebic abscess should be differentiated from pyogenic abscess, hepatic tumors and hydatid cysts. **So C/P and lab investigations should always be correlated to U/S and CT findings.**



Abdominal CT scan demonstrating a large abscess in the right hepatic lobe

Treatment

Medical Treatment (Primary treatment) → highly successful

- **Metronidazole:** Drug of choice = (Metronidazole).
 - 800 mg 3 times daily for 7 – 10 days.

Surgical Treatment

▪ Aspiration:

- Indications:
 1. Resistant after medical treatment (within 72 hours).
 2. Large abscess.
 3. Rupture.
 4. Patient is very toxic.
- Precautions:
 1. Under guide of U/S.
 2. Under cover of Metronidazole.
 3. After full aspiration inject:
 - Metronidazole 500 mg.
- Technique:
 1. Aspiration is done using wide bore (1-2mm) spinal needle (under local anesthesia).
 2. Site of aspiration:
 - In anterior Abscess → below the costal margin ant.
 - In posterior Abscess → in 10th intercostal space post.
 3. U/S is repeated few days later (to detect recollection).

▪ Open drainage

- Indications:
 1. Failure of aspiration (thick contents, multilocular).
 2. Rapid refilling, or rupture leading to peritonitis.
 3. Secondary infection.
 4. Abscess in the Lt. lobe (for fear of rupture in pericardium).
- Technique:
 1. Incision:
 - Rt. lobe → extra-pleural extra-peritoneal through bed of 12th rib.
 - Lt. lobe → Midline.

Prognosis

- Mortality rate in uncomplicated cases is <5%[□].

3- Pyogenic Liver Abscess

Incidence

Predisposing factors

- Old age.
- DM.
- Immunocompromised state.
- Pre-existing liver lesion: as hydatid cyst, amoebic abscess.

- ▶ **Etiology:** Ascending cholangitis(35-50%)by E.coli, portal pyemia, septicaemia, direct spread, 2ry inf.
- ▶ **C/P:** c/p of the cause + as acute cholecystitis
- ▶ **Comp.:** direct spread, rupture
- ▶ **Invest.:** CBC, U/S & CT, guided aspiration(C&S)
- ▶ **TTT:** A-If multiple small abscesses : antibiotics
B- If large: (>4cm): Percutaneous or open

Site

- Right > left lobe[□].
- Multiple abscesses are suggestive of phlebitis, suppurative cholangitis, and septicaemia[□].

Organism

- Gram -ve aerobic rods, streptococci, and anaerobes.

Main Routes

Bile Duct

- **Ascending cholangitis:** (most common[□] 35-50% of cases)
 - The likely organism is E.coli or other gram -ve organism.
 - Stasis of bile.
 - Secondary to stone or stricture.

Portal Vein

- **Portal pyemia:** (streptococci and anaerobes are the commonest organisms)
 - a) Acute Suppurative appendicitis.
 - b) Acute Suppurative diverticulitis.
 - c) Infected thrombosed piles.
 - d) Infected carcinoma of the colon.
- **Neonatal umbilical sepsis:**
 - Infected umbilical stump → umbilical vein or Para umbilical v. → portal vein.
 - Occurs in the newborn in exchange transfusion.

Hepatic Arteries

- **Septicaemia.** (staphylococcus aureus is the usual causative organism)
- **Systemic pyemia:** secondary to
 - Infective endocarditis.
 - Acute osteomyelitis.

Direct Extension

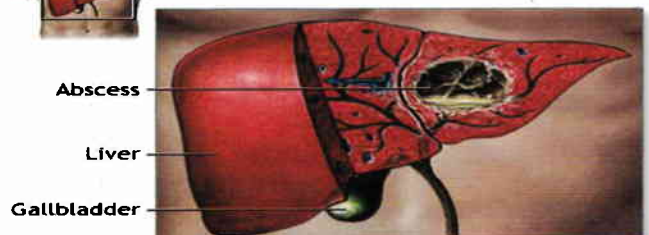
- Sub-phrenic abscess.
- Empyema thoracis.
- Penetrating wound.
- Suppurative cholecystitis.

2ry Infection of

- Hydatid cyst.
- Amoebic liver abscess.
- Tumour.



pyogenic liver abscess



Clinical Picture

C/P of the primary cause + C/P like those of acute cholecystitis

(fever, malaise, RT upper quadrant pain)

Complications

- Direct extension to surroundings:
 - Pleura, lung, pericardium & peritoneum.
- Rupture (as amoebiasis).

Investigations

1. Laboratory:

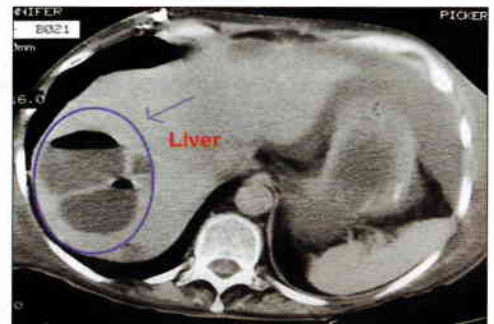
- CBC:
 - Leucocytosis (but may be absent), anemia & high ESR.
- LFTs:
 - Transaminases & alkaline phosphatase.
 - Serum bilirubin (in case of cholangitis).
 - Serum albumin level rapidly falls.

2. Radiological:

- U/S & CT:
 - Detects number, site & size of abscess.

3. Instrumental:

- Guided aspiration to confirm the diagnosis.
- The aspirated material is sent to C&S.



Treatment

1. **If multiple small abscesses** → broad spectrum antibiotics (amino-glycosides, clindamycin, or metronidazole).
2. **If large: (>4cm)**
 - A. Percutaneous drainage guided by U/S ☑ (under local anesthesia) & tube drain is inserted (until discharge stops). "blind aspiration may go through pleura → empyema"
 - B. Open surgical drainage (if percutaneous drainage fails due to thick pus or multiloculated abscess) drain is left in position until drainage ceases.
3. **Treatment of the cause.**

Treatment of amoebic abscess is primarily medical in contrast to pyogenic abscess ☑.

Prognosis

- Mortality (more than 18-20% ☑) is largely diagnosed by the underlying cause.

Liver Tumors

Benign Tumors

- 1- Adenoma.
- 2- Hemangioma.
- 3- Focal nodular hyperplasia.
- 4- Hamartoma.

Malignant Tumors

A. Primary tumors:

- Hepatocellular carcinoma (HCC).
- Cholangiocarcinoma.
- Cholangiohepatoma.
- Hepatoblastoma (D.D. Neuroblastoma, Wilm's tumor).

B. Secondary tumors: Metastasis.

Benign Liver Tumors

1 - Adenoma

Definition

- They are rare benign liver tumors.
- True neoplasm which predispose to complications and should usually be resected.

- ▶ **Etiology:** A rare benign tumor in ♀ using OCPs
- ▶ **Path.:** Usually multiple
- ▶ **C/P:** Asymptomatic, swelling, pain
- ▶ **Comp.:** hepatoma, rupture, peritonitis
- ▶ **Invest.:** 1- CT (**the best**) 3- Biopsy
 2- α-fetoprotein (to exclude hepatoma)
- ▶ **TTT:** 1- Stop OCPs 2- Localized resection

Age & sex

- Females in menstrual age (Especially those on contraceptive pills[□]).
- Growth is sex hormone sensitive → OCP's increase growth.

Pathology

Macroscopic Picture

- Soft well circumscribed light yellow fleshy tumor with vessels over it.
- Frequently multiple².

Microscopic Picture

- Sheets of mature hepatocytes.
- No portal triads & no central vein.
- Mimic nodular hyperplasia of cirrhosis ☒.

Clinical Picture

1. **Asymptomatic** & accidentally discovered during routine U/S.
2. **Swelling** in Rt. hypochondrium.
3. **Pain** is due to stretch of the liver capsule.

Complications *(Very rare)*

- 1- Malignant transformation → hepatoma.
- 2- Spontaneous rupture → internal hemorrhage.
- 3- Clinical picture of generalized peritonitis.

Investigations

1. CT (triphasic) well circumscribed vascular solid tumor with peripheral arterializations in normal liver.
2. Abdominal U/S, and angiography.
3. Liver function test → normal.
4. α -fetoprotein → normal (to exclude hepatoma).
5. Biopsy: for confirmation of the nature of the lesion as there is no radiological findings to differentiate between this lesion & malignant tumors

Treatment

1. **Stop the contraceptive pills** (If < 2cm) → Spontaneous regression.
2. **Surgical → Localized resection.**
 - Indicated if there is doubt of malignancy or large symptomatic cases.

2- Hemangioma

1. **Most common** benign tumour of the liver (Hamartoma that usually requires no treatment).
2. The liver is the most common visceral organ affected by haemangioma.
3. Usually **cavernous**.

Clinical Picture

1. Asymptomatic.
2. Pain.
3. Rupture & intraperitoneal hemorrhage.
4. Heart failure in infant (A-V fistula).

Investigations

- **U/S:**
 - Focal lesion (hyperechoic).
- **Angiography or CT with contrast:**
 - Are diagnostic.

Treatment

- **Asymptomatic & small** → follow up.
- **Excision** : if there is pain or bleeding

hepatic haemangioma



3- Focal Nodular Hyperplasia (FNH)

Definition

- Unusual benign condition in which there is focal overgrowth of functioning liver tissue supported by fibrous stroma.

Etiology

- Unknown etiology but may be due to contraceptive pills.

OCP → adenoma, FNH

Macroscopic

- Nodular, firm, paler than the surrounding, stellate scar.



Microscopic

- Central scar, fibrous septa.

Investigations

- **CT:**
 - Can detect pathognomonic stellate scar, vascular lesion.
- **Sulphar colloid scan** **differentiate** between FNH from either a benign adenoma and 1ry or metastatic cancer

Treatment

It is not a true neoplasm

- No treatment is required in most cases.
- Discontinuation of contraceptive pills.
- If suspecting malignancy, exploration and frozen section are recommended.

Malignant Liver Tumors

I-Hepatocellular carcinoma (hepatoma)

Incidence

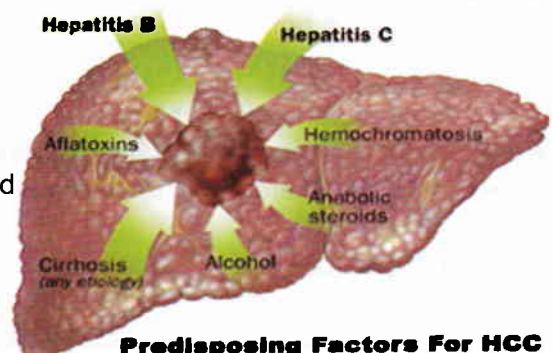
- **HCC:**
 - The first cause of death from cancer world wide.
 - More in old males.
 - In Egypt → metastasis liver cancer
 - Highest prevalence in the Far East → hepatoma

Predisposing Factors

(90% of cases are related to HCV, HBV & liver cirrhosis)

- **Hepatocellular carcinoma:**
 1. Hepatitis B & C.
 2. Liver cirrhosis of any cause (except Wilson's disease) e.g. post hepatic, alcoholic & hemochromatosis (10% of patients → HCC).
 3. **Carcinogenic material:**
 - a- Aflatoxins^{LD} → grains contaminated with Aspergillus flavus.
 4. **In children:**
 - a- Biliary atresia.
 - b- α 1-antitrypsin deficiency.
 - c- Galactosemia.
 5. **Adenoma:** (Rare).
 6. Association of HCC with use of contraceptive pills and anabolic steroids has been suggested but still weak.

- ▶ 1st cause of death from cancer world wide
- ▶ **PDF:** Hepatitis B&C, Liver cirrhosis, aflatoxins
- ▶ **C/P:** rapid deterioration of cirrhotic patient
 - **hepatic:** H/O of cirrhosis, exacerbation
 - **extrahepatic:** paramalignant \$
- ▶ **Invest.:** imaging (U/S, CT, MRI), markers ($\uparrow$ α -feto protein), hepatic angiography
- ▶ **TTT:** - Inoperable: chemo, ligation, ethanol,....
- Operable: formal hepatic resection



Predisposing Factors For HCC

Mechanism of hepatoma in:

- Hepatitis B:** integration between DNA of virus & DNA of hepatocytes → abnormal proliferation of hepatocytes (**More precancerous**).
- Hepatitis C:** no integration because hepatitis C is RNA virus, hepatoma occurs due to cirrhosis (by chronic irritation).

Pathology

▪ **Hepatocellular carcinoma:**

- **Macroscopic:**

▪ Mass infiltrating edge + area of hemorrhage and necrosis it may be:

1. **Massive form** → Single large mass surrounded by satellites
2. **Nodular form** → Multiple nodules scattered over the liver
3. **Diffuse form** → Fine nodules involve whole liver.

- **Microscopic:**

- Malignant hepatocytes (adenocarcinoma) with:
 - Pleomorphism & mitotic figures.
 - Acidophilic cytoplasm and acidophilic nucleoli.
- High vascularity derived from **hepatic artery** (not from the portal vein).

Fibrolamellar type

- It is a variety of massive form.
- Affects non-cirrhotic young females.
- Better prognosis
- Microscopically:
 - Multiple fibrous septa make it resembles FNH.

Spread

Direct

- To stomach, colon, kidney & diaphragm.

Lymphatic

- To LNs in porta hepatis → celiac LNs → thoracic duct → Lt. supra-clavicular LNs "Virchow's LN."

Blood

- To Lung & bones.

Clinical Picture of Liver Tumors

- Rapid deterioration of health of a patient with liver cirrhosis → Suspect hepatoma[☑].

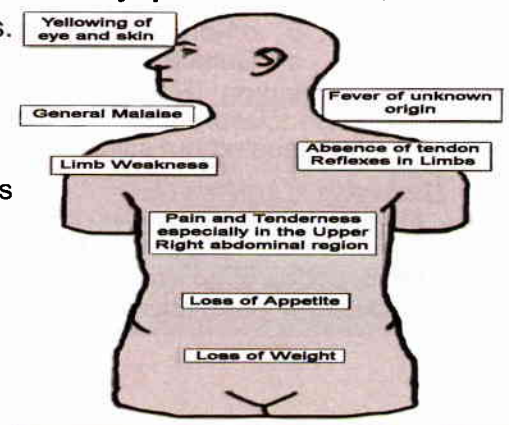
Frank (Hepatic) Presentations

- In areas where HCC is predominant, an early detection program is advised by U/S & alpha fetoprotein

▪ **Symptoms:**

1. History of chronic hepatitis & liver cirrhosis.
2. Exaggeration of pre-existing hepatic condition[☑]:
 - a. Jaundice.
 - b. LCF → hepatic encephalopathy.
 - c. Portal hypertension → hematemesis & resistant ascites.
3. Swelling → (Late) localized to liver.
4. Pain → (Late) dull aching in upper abdomen.
5. Hepatocellular carcinoma in black Africans may resemble amoebic hepatitis with pain and fever.

Symptoms of Liver Cancer



May cause Hemoperitoneum in 10% of cases

▪ **Signs:**

1. **General:**

- a. Jaundice, fever and cachexia.
- b. Enlarged supra-clavicular L.N. (Lt. & Rt.).

2. **Local:**

1. Liver is enlarged, irregular & tender.
2. Localized tender mass may be felt (in 50%).
3. Ascites.
4. Friction rub, arterial bruit (30%).
5. Occasionally murmur over the tumor (Mamoun sign[☑]).

Occult (Extra-hepatic) Presentations

▪ **Non-metastatic (Paramalignant syndrome[☑]):**

1. Hypoglycemia due to Insulin-Like Polypeptide(ILP).
2. Polycythemia (erythropoietin).
3. Hypercalcemia & pathological fracture (PTH like)
4. Cushing like (ACTH) → (no moon face due to cachexia, diagnosed by HTN, DM, hypokalemia & pigmentation).
5. Hypotension (VD) or Hypertension (ACTH)
6. Fever (FUO).

Investigations

I. **For diagnosis:**

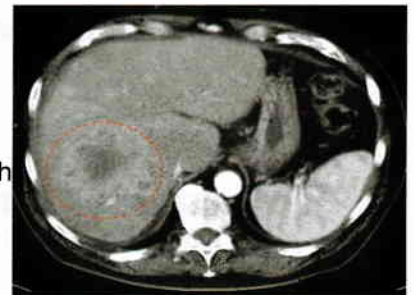
▪ **U/S, CT or MRI:**

- Detect the site and extent of tumor
- U/S is the screening investigation of choice in HCC for high risk population although it is not very sensitive for liver tumors especially in cirrhotic liver but it is of low cost.

▪ **Tumor markers:**

1. **Carboxy prothrombin:**
 - More specific.
2. **Alpha fetoprotein[☑]:** (elevated in 55-95% of patients)
 - Useful tumor marker for follow up although its low sensitivity..
 - Normal level 0 – 10 ng / dl.
 - 500 ng / dl → high suspicion of malignancy.
 - Test of choice to exclude onset of hepatoma in a cirrhotic patient[☑].
 - Level above 200 ng/dl is diagnostic of HCC

- **Hepatic angiography:** (through hepatic artery as most of the 1ry and metastatic tumors of the liver take their blood from hepatic artery[☑])
 - Shows characteristic high vascularity.



- Percutaneous guided needle biopsy (by U / S or CT):(Provided that PT is normal)
 - Point of controversy.
 - Done only in inoperable cases before chemotherapy.
- Laparoscopy: can give us direct biopsy.

2. For staging:

▪ Chest X-ray and CT:

- For search for lung metastasis.

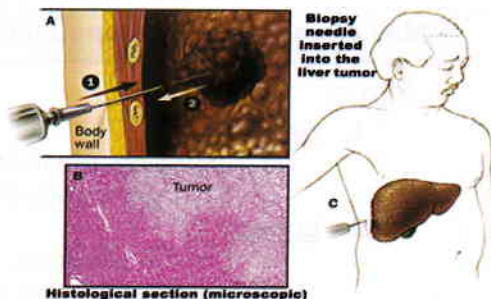
3. For preoperative preparation:

▪ LFTs:

1. ↑ serum bilirubin.
2. ↑ serum alkaline phosphatase.
3. Other tests show pattern of cirrhosis.

▪ Others:

1. CBC: anemia or Polycythemia.
2. KFTs: Renal failure due to hepato-renal association.
3. Fasting blood sugar: ↓ due to insulin -like hormone.



Treatment

- Liver resection or transplantation are the only hope for cure but only small proportion of cases is suitable for surgery so preoperative assessment **by Child's classification and size, site of the tumor** is of a great help in evaluation of the prognosis of surgery.

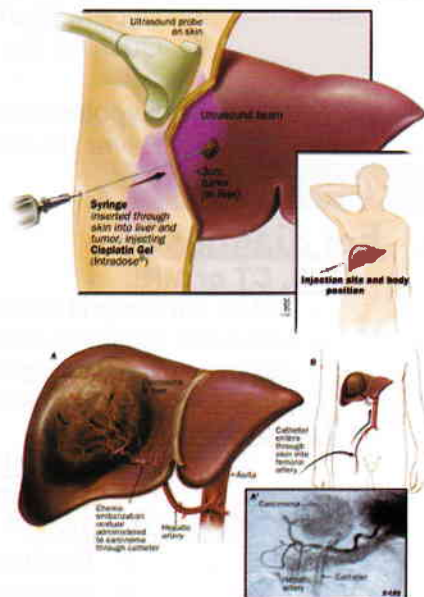
Inoperable Cases

1. Systemic chemotherapy.

2. Ligation of the feeding artery.

3. Transarterial Chemoembolization (TACE):

- Chemotherapeutic agent is loaded on gel foam and is injected by angiographic techniques into the hepatic artery, the artery is blocked → tumor ischemia and the drug is selectively delivered to its target.



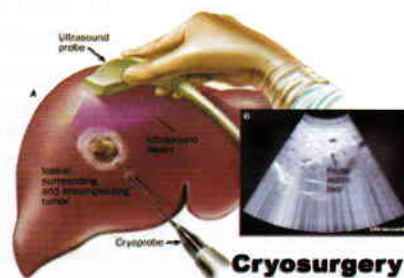
4. Cryosurgery.

5. Percutaneous ethanol injection in the tumor

6. Thermal ablation by:

- Radio-frequency:
 - Percutaneous.
 - Open or laparoscopic.
- Laser therapy.

7. Monoclonal antibody therapy.



(Anatomical (formal) hepatic resection)

Operable Cases

- The ideal treatment for a tumor that is localized to one liver lobe is to resect it rather than transplantation provided that the liver is non-cirrhotic(RT. or LT. hemihepatectomy).
- The presence of cirrhosis (this indicate transplantation[☑]) in most cases poses some problems due to:
 - 2- Bleeding tendency.
 - 3- Poor function of the remaining part of liver.
 - 4- Diminished capacity of the liver cells to regenerate.

II-Liver Metastasis

- It is the **most common** malignant tumor of the liver (20 times more common than 1ry malignancies)[☑].
- **The commonest primary sites are:**
 - a. Stomach, breast, kidney.
 - b. Colorectal, lung.
 - c. Pancreas, ovary.
 - d. Melanoma.
 - e. 2ry carcinoid tumors from small intestine or bronchus.
- **The metastasis reaches the liver via:**
 - a. **Portal vein:** (The commonest route)
→ e.g. Carcinoma of colon, rectum, pancreas & stomach.
 - b. **Hepatic artery:**
→ e.g. Carcinoma of lung, breast, kidney, uterus & ovary.
 - c. **Lymphatics → breast.**
 - d. **Direct extension:** → E.g. tumours of G.B., stomach & colon.

- ▶ commonest malignant liver tumor in Egypt
- ▶ **Portal vein** is The commonest route from GIT
- ▶ **C/P:** Picture of primary tumor +
Hepatomegaly, Ascites & Jaundice.
- ▶ **Invest.:** LFTs, markers, imaging (U/S, CT)
- ▶ **TTT:** - non-resectable: chemotherapy, monoclonal antibodies, embolization
- Resectable: resection if < 3 liver metastases

Pathology

- They are multiple, umbilicated nodules due to central necrosis.
- They are usually adenocarcinomas
- They may be discovered at the same time of the primary tumor (synchronous) or months or years later after resection (metasynchronous).
- Over 90% of cases have tumor deposits in other organs

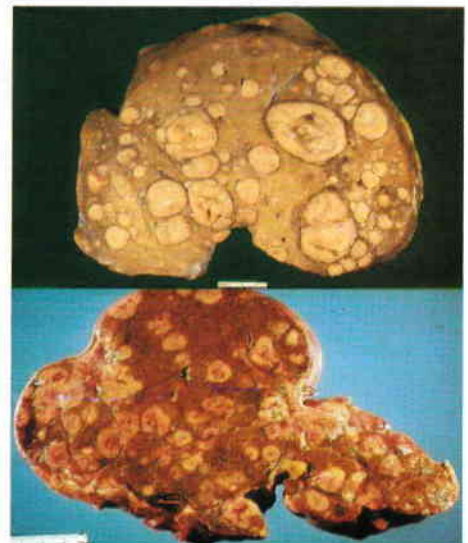
Clinical Picture

- Picture of primary tumor +
- Hepatomegaly, ascites and jaundice.

Investigations

I. Laboratory:

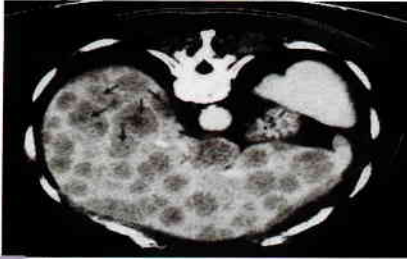
- **CBC:** anemia
- **LFTs:**
 - ↑ Alkaline phosphatase
 - ↑ serum bilirubin.
- **Tumor markers.**



2. Radiological:

▪ U/S, CT or MRI and PET-scan:

- Are diagnostic and show the number and sites of metastases.



Treatment

Once liver metastasis has occurred, the tumor is inoperable except for some cases of colorectal tumors (max. no is 3) ☐

Treatment of non-resectable tumors

1. **Chemotherapy:**

- a- **Systemic** → If other organs are involved.
- b- **Local** (intra-arterial injection) → if liver is the only site of metastasis.

2. **Monoclonal antibodies.**

4. **Sectorial Portal vein embolization** (atrophy of the affected segment).

Surgical Resection

(Role of resection in colorectal metastasis is well established 5 year survival rate 35%)

▪ Prerequisites:

- 1- Completely resectable 1^{ry} tumor.
- 2- Solitary liver metastasis or multiple (<4) confined to one surgical lobe.
- 3- Liver is the only affected organ with metastasis.

Liver metastasis usually occurs on top of normal liver, so patient can tolerate up to 60 – 70 % of liver resection

Causes of Hepatomegaly

1. Liver congestion due to:

- a) Rt. Ventricular failure.
- b) Tricuspid incompetence or stenosis.
- c) Pericardial effusion and constrictive pericarditis.
- d) Inferior vena caval obstruction.
- e) Budd-Chiari syndrome.
 - Classical triad of abdominal pain, ascites and hepatomegaly ☐.
 - Caudate lobe hypertrophy is often present ☐.
 - Treatment: liver transplantation ☐.
- f) Veno-occlusive disease.

2. Infections:

- a) Viral (viral hepatitis, EBV, CMV, HSV).
- b) Bacterial (Pyæmic abscess, brucellosis, typhoid, syphilis).
- c) Protozoal (malaria, amoebic liver abscess, Kala Azar).
- d) Helminthes (Bilharzias, Hydatid cyst).

3. Tumors (Iry & 2ry).**4. Obstructive jaundice.****5. Blood diseases:**

- a) Leukemia, lymphoma.
- b) Hemolytic & megaloblastic anemia.

6. Metabolic disorders:

- a) Fatty liver, amyloidosis.
- b) Lipid storage disorders e.g. Gaucher's disease.
- c) Glycogen storage disorders e.g. Von Gierke's disease.

Causes of Enlarged Tender Liver

- 1- Viral hepatitis.
- 2- Amoebic hepatitis.
- 3- Hepatoma.
- 4- Congested liver (CHF).

Causes of Nodular Liver

- 1. Bilharziasis.
- 2. Post-necrotic.
- 3. Malignancy.
- 4. Syphilitic gumma.
- 5. Hydatid cyst.

DD of Liver Cyst

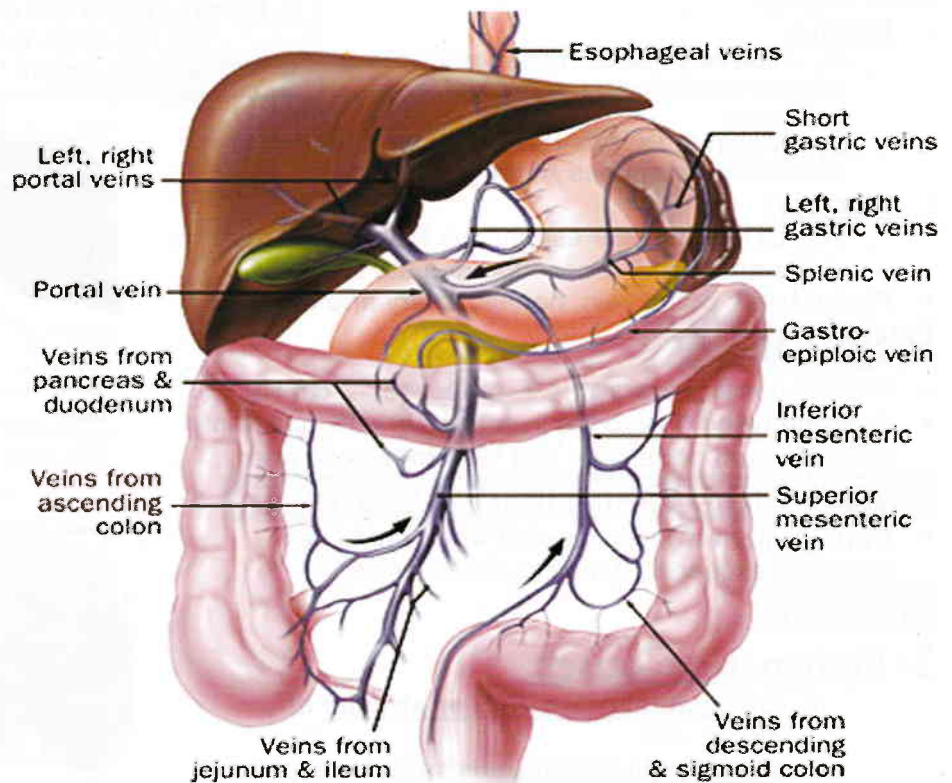
A. Parasitic:

- Hydatid cyst.

B. Non-parasitic:

- 1) Choledochal cyst.
- 2) Hemangioma.
- 3) Polycystic liver.
- 4) Retention cyst.
- 5) Degenerated cyst.
- 6) Traumatic blood cyst.

CHAPETR 9



PORTAL HYPERTENSION

Introduction

Anatomy

Portal vein:

(Valveless vein that provides 75% of hepatic blood supply)

Begins:

- By union of superior mesenteric vein & splenic vein behind the neck of pancreas.
- By division to Rt. (short oblique) & Lt. (longer transverse) branches At the porta hepatis

It drains:

- The GIT from lower esophagus to the upper part of the anal canal (above the dentate line).

Portal triad: portal vein, hepatic artery and bile duct.

Physiology

- Normal portal pressure = 7 mm Hg (8-12 cm H₂O).
- Average blood flow to the liver is 1500 ml / minute of which $\frac{2}{3}$ from portal vein and $\frac{1}{3}$ from hepatic artery.

Definition of Portal Hypertension

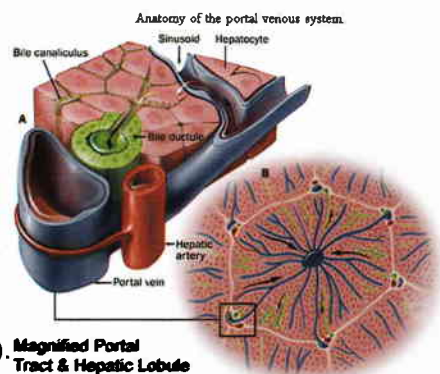
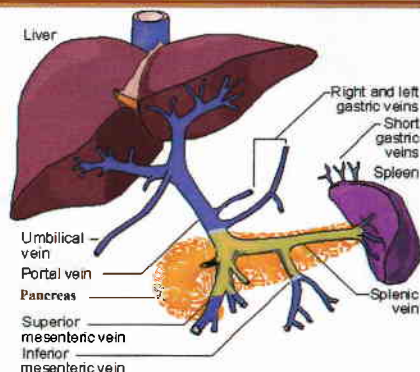
- Portal hypertension is present when portal vein pressure exceeds 20 mmHg (25-30 cmH₂O).

Etiology

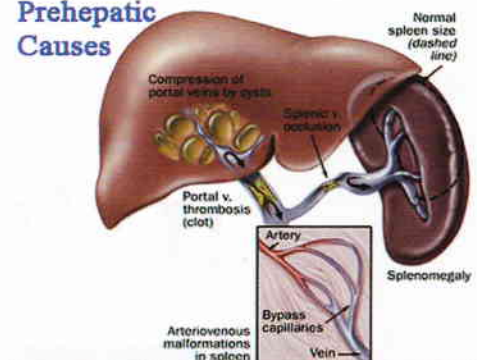
I- Prehepatic Causes:

1. **Congenital narrowing or atresia:** of portal vein.
2. **Portal vein thrombosis** secondary to:
 - Neonatal umbilical sepsis.
 - Intrabdominal sepsis (e.g. severe appendicitis).
 - Viscosity as polycythemia, dehydration, contraceptive pills
3. **Invasion by malignancy:** e.g. HCC, pancreatic carcinoma.
4. **Compression of portal vein by cyst.**
5. **Arterio-venous malformation.**

- ▶ **Def.:** portal v. pressure > 25 mmHg
- ▶ **Etiology:** prehepatic, hepatic, suprahepatic
- ▶ **C/P:** - Elective: portal HTN, LCF, cause
 - Emergency: bleeding esoph. varices, encephalopathy
- ▶ **Invest.:** Upper GIT endoscopy, U/S, duplex, for cause & comp.
- ▶ **TTT:** elective, urgent



Prehepatic Causes

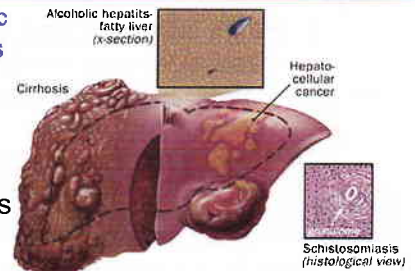


II- Intrahepatic Causes:

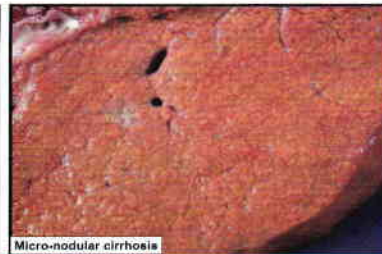
1- Pre-sinusoidal:

- a- Bilharzial periportal fibrosis.
- b- Congenital fibrosis of portal tracts.
- c- Infiltration of portal tracts by abnormal cells e.g. Hodgkin's lymphoma, leukemias and sarcoidosis

Hepatic Causes



Macronodular cirrhosis



Micronodular cirrhosis



Biliary cirrhosis

2- Sinusoidal:

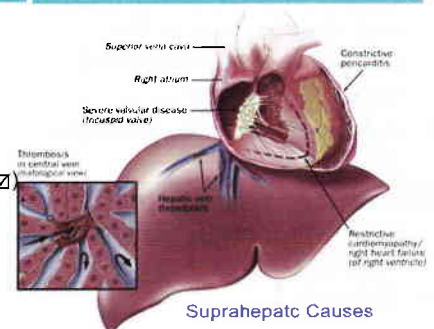
- Liver cirrhosis.

3- Post-sinusoidal:

- Veno-occlusive disease.

III- Suprahepatic Causes:

- 1- Budd-chiari syndrome (occlusion at hepatic vein) ✓
- 2- High IVC obstruction.
- 3- Constrictive pericarditis and pericardial effusion.
- 4- RVF.
- 5- Tricuspid stenosis or incompetence.



Suprahepatic Causes

1. **Most of cases of portal hypertension in adults are due to:** liver cirrhosis and Bilharzial hepatic fibrosis.
2. **Liver cirrhosis causes portal hypertension through:**
 - a- Obliteration and compression of sinusoids.
 - b- Compression of radicals of portal and hepatic veins by the excessive fibrosis.
 - c- Development of multiple A-V shunts between branches of hepatic artery and those of portal vein.
3. **Veno-occlusive disease:**
 - Post-sinusoidal obliteration of hepatic venules due to hypersensitivity reaction against plant toxins used in herbal medicine (in children).
4. **Budd-chiari syndrome** ✓:
 - Characterized by: impaired liver function, pH⁺
 - Obstruction of hepatic veins by either:
 - a- Thrombosis (spontaneous or 2ry to polycythemia or use of contraceptive pills).
 - b- Malignant invasion.
 - Small cirrhotic liver with enlarged caudate lobe demands exclusion of Budd-chiari syndrome.

5. **PV thrombosis and schistosomiasis** may cause increase in PV pressure while hepatic venous wedge pressure is normal.
6. Spleen is the capacitance of portal circulation while liver is the capacitance of systemic circulation.
7. There is no portal hypertension without splenomegaly.

Pathology of Portal Hypertension

I. Portosystemic Anastomosis:

- They are normally collapsed due to lower pressure in the portal circulation
- In portal hypertension, they become engorged in attempt to divert blood away from the portal circulation.
- **The important sites of these collaterals are:**

Site	Between	
	Portal circulation	Systemic circulation
1- Lower end of esophagus and the fundus of the stomach (esophageal & fundic varices) ☑	Esophageal veins from Left gastric vein (coronary vein) & short gastric veins	Esophageal veins from Azygous & hemiazygous veins
2- Around umbilicus (Caput Medusae) ☑	Paraumbilical vein	Superior and inferior Epigastric veins
3- Lower end of the rectum and Anal canal (anorectal varices) ☑	Superior rectal veins	Middle & inferior rectal veins
4- Others ➔ Retroperitoneal space	Superior and inferior mesenteric veins	Posterior abdominal wall veins and subdiaphragmatic veins

- Anastomosis between portal and systemic circulation doesn't occur at peritoneal covering of liver ☑.
- In splenic vein thrombosis they develop around fundus of the stomach ☑.

2. Splenomegaly:

➤ Causes:

1- Congestive Splenomegaly:

- Secondary to progressive to portal hypertension.
- This leads to:
 - A- Hypersplenism.
 - B- Recurrent attacks of splenic infarctions: causing severe stitching pain and adhesions with the diaphragm and anterior abdominal wall.

2- RES hyperplasia:

- Due to absorption of Bilharzial toxins.

Spleen is the capacitance of portal circulation



Splenomegaly on laparoscopy

3- Ascites:

➤ Causes: (It is multifactorial):

1. Hypoalbuminemia:
 - If associated liver cell failure.



There is no ascites without hypoalbuminemia, portal hypertension is a localizing factor

2. Portal hypertension:

- It only localizes the filtration of fluid into the peritoneal cavity.

As portal hypertension is a localizing factor, so ascites occurs 1st of all → L.L. edema (Ascites precox)

3. Salt and water retention due to:

- ↑ Production of ADH and aldosterone due to Hypovolemia.
- ↓ Destruction of ADH and aldosterone by the liver.

4. Lymphorrhea (weeping liver):

- Postsinusoidal obstruction → ↑ sinusoidal pressure → ↑ lymph production, which extravasates into the peritoneal cavity (it occurs mainly in Lannec's cirrhosis).

4- Congestion of the Whole GIT: congestive gastropathy & enteropathy.

5- Liver: (according to the cause of portal hypertension)

1. Prehepatic causes:

- The liver is usually normal.

2. Post-hepatic causes:

- The liver is enlarged, soft and tender.

3. Hepatic causes (mainly cirrhosis and bilharziasis):

- At first there is hepatomegaly then becomes shrunken, firm with a sharp border.

Clinical Presentation

Elective Cases

➤ The patient may present with:

- i- Manifestations of portal hypertension:
 - 1- C/P of Porto-systemic anastomosis.
 - 2- C/P of splenomegaly
 - 3- C/P of congestive gastropathy.
- ii- Manifestations of liver cell failure.
- iii- Manifestations of the cause.

Emergency Cases

➤ The patient may present to the ER by one of the following complications:

- 1- Active bleeding from esophageal varices (hematemesis and / or melena)
- 2- Hepatic encephalopathy.

Elective Cases

I- Manifestations of portal hypertension:

1- Clinical picture of porto-systemic anastomosis:

a. Esophageal varices:

➤ Present by:

- Past history of hematemesis, melena &/or bleeding per rectum (if massive).
- 2\3 of patients develop varices, 2\3 of them subsequently experience variceal hemorrhage.



b. Caput medusa:

➤ Presents by:

- Dilated veins around the umbilicus.
- Palpable thrill over it.
- Auscultated venous hum due to ↑ blood flow.

c. Anorectal varices (rare):

- Hemorrhoids in patients with portal hypertension is usually primary.

2- Clinical picture of Splenomegaly:

➤ Symptoms:

- 1- Abdominal enlargement.
- 2- Abdominal pain:
 - Dull aching (stretch of splenic capsule).
 - Dragging (pulling on the ligament).
 - Stitching (perisplenitis).

Perisplenitis:

- Is due to recurrent attacks of splenic infarction → healing by fibrous tissue → adhesions between the spleen and the diaphragm and ant. abdominal wall.

➤ Signs:

- 1- Firm mass in Lt. hypochondrium.
- 2- Preserved notch.

➤ Complications:

Secondary hypersplenism:

- Due to Splenomegaly & portal hypertension.
- It leads to thrombocytopenia, less commonly leucopenia and rarely anemia.



3- Clinical picture of congestive gastropathy:

- Anorexia, dyspepsia, indigestion and malabsorption.

Causes of dyspepsia:

- 1- Gastric congestion.
 - 2- Peptic ulcer in 30% of cases due to inactivation of gastrin hormone.
- Pressure on the stomach by an enlarged spleen (is a wrong belief).

II- Manifestations of Liver Cell Failure:

1- General:

- Loss of weight, weakness, low grade fever, jaundice, foetor hepaticus and lower limb edema.

2- Ascites:

➤ Symptoms:

- 1- Abdominal enlargement.
- 2- Abdominal discomfort.
- 3- Dyspepsia & dyspnea.

➤ Signs:

- 1- Fullness of both flanks.
- 2- Bilateral shifting dullness.

3- Skin manifestations:

- Palmar erythema, spider naevi, paper-money skin and white nails.

4- Endocrinal manifestations:

- Male: Gynecomastia, feminine hair distribution, ↓ Libido, impotence and testicular atrophy
- Female: breast atrophy, ↓ Libido, amenorrhea and infertility.

5- CVS:

- Hyperdynamic circulation (collapsing pulse, wide pulse pressure)
- Cyanosis and clubbing.

6- Hematological manifestations:

- Anemia and bleeding tendency.

7- Hepatorenal failure.

8- Hepatic encephalopathy: some responsible toxins for encephalopathy are ammonia, methionine and short chain fatty acids[□].

Risk factors for hepatic encephalopathy

- 1- GIT bleeding.
- 2- Infection.
- 3- Increased dietary protein.
- 4- Constipation.
- 5- Drugs: sedatives, antidepressants & diuretics
- 6- Hypokalemia.
- 7- Paracentesis (volume > 3 – 5 liters).
- 8- Porto-systemic shunts.

III- Manifestations of the Cause:

1 - Bilharziasis:

- Anemia, easy fatigability.
- Terminal hematuria, dysentery.

2- Chronic liver disease: *(according to the cause)*

➤ Post viral:

- There may be a history of acute viral hepatitis (or might present only with history of previous blood transfusion).

➤ Lannec's cirrhosis:

- High & continuous alcoholic consumption.

➤ Biliary Cirrhosis:

- History of long standing obstructive jaundice.

➤ Cardiac cirrhosis:

- History of long-standing Rt. Sided heart failure.

- Congenital causes: e.g.
 - **Wilson's disease:** 5 - 30 years child with liver cirrhosis, basal ganglion affection, renal tubular damage and Kayser Fleisher rings.
 - **Hemochromatosis:** male 40 years with liver cirrhosis, DM, with dark pigmentation
 - **α -1 antitrypsin deficiency:** liver cirrhosis and emphysema.

Complications

- 1- Variceal bleeding (esophageal, gastric, others are rare.)
- 2- Congestive gastropathy.
- 3- Hypersplenism.
- 4- Ascites.
- 5- Renal failure.
- 6- Hepatic encephalopathy.
- 7- Metabolic alkalosis.

Investigations

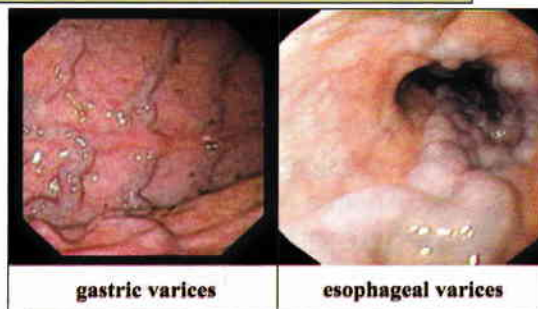
1- For diagnosis:

- **Fiberoptic upper endoscope:**

- Detection of esophageal varices:

(Grading of esophageal varices)

- Grade I: just elevation of mucosa by dilated veins.
- Grade II: elevated tortuous veins with normal mucosa in-between.
- Grade III: elevated tortuous veins with abnormal mucosa in-between
- Grade IV: erosion, ulceration and shows cherry red spots i.e. impending rupture.



Impending bleeding is considered if pressure > 35 cm H₂O or in the presence of cherry red spots. ☑

- **Detection of portal hypertension:**

- **Duplex scan:** Pressure & patency of portal vein.
- **Abdominal U/S:**
 - It may show:
 - Liver Cirrhosis, splenomegaly, ascites, portal vein diameter (>14mm in case of portal HTN), hepatoma.

- **Barium swallow** (not used now):

- Can visualize varices in 90% of cases as multiple, smooth, rounded filling defects (grape-like or honey-comb appearance).

2- For the cause:

- **Urine and stool analysis:** for bilharzial ova
- **Sigmoidoscopy:**
 - To visualize bilharzial lesions and take biopsy snips.
- **Cystoscopy:**
 - To visualize bilharzial lesions
- **Hepatitis markers.**
- **Liver biopsy after assessment of PT and PC.**

3- **For complications::**I- **KFTs:** To exclude hepatorenal failure.II- **LFTs:**

- Serum Albumin: if $< 2.5 \text{ gm\%}$ → ascites.
- PT & concentration: are disturbed, this test is the most sensitive LFTs.
- Serum bilirubin: may be increase (mainly direct).
- ALT & AST: elevated in late stages.

III- **For detection of hypersplenism:**

- **CBC**: Anemia, leucopenia, thrombocytopenia, pancytopenia.
- **BM examination** → hypercellularity.
- **Radioactive isotope studies**:
 - ⇒ By labeling the patient RBCs with Cr^{51} → ↓ 1/2 life & radioactivity of spleen is increased.

Treatment

Elective	Urgent
- Silent varices. - Past history of bleeding esophageal varices. - Ascites.	- Bleeding esophageal varices. - Encephalopathy.

1- Management Of Actively Bleeding Esophageal Varices**Presentation**

1. **Variable degrees of shock.**
2. **Hematemesis** → Vomiting of coffee ground material (acid hematin formation) due to bleeding above the ligament or Trietz. (It may be red blood if severe bleeding)
3. **Melena** → Passage of soft tarry offensive stool (It may be red blood if severe bleeding due to GI hurry)
4. **Picture of the cause:**
 - a- Bilharziasis → history of bilharziasis, anemia, easy fatigability & HSM
 - b- Cirrhosis → Jaundice, encephalopathy, shrunken liver & splenomegaly.

- ▶ **C/P**: Shock, Hematemesis, Melena + c/p of the cause (Bilharziasis, Cirrhosis).
- ▶ **Inv.:** Upper GIT endoscopy
- ▶ **General principle of TTT:**
 - a) Hospitalization
 - b) Resuscitation By fresh blood transfusion. and monitoring
 - c) Prevent encephalopathy by gastric lavage, enema, lactulose & Metronidazole
 - d) Stop bleeding by upper GI endoscopy + injection sclerotherapy, drugs, balloon tamponade, TIPPS if all measures failed

Investigation of choice

- Upper GIT endoscopy.

Treatment

A- Hospitalization:

Even minor hematemesis may be followed by massive bleeding.

B- Resuscitation and monitoring:

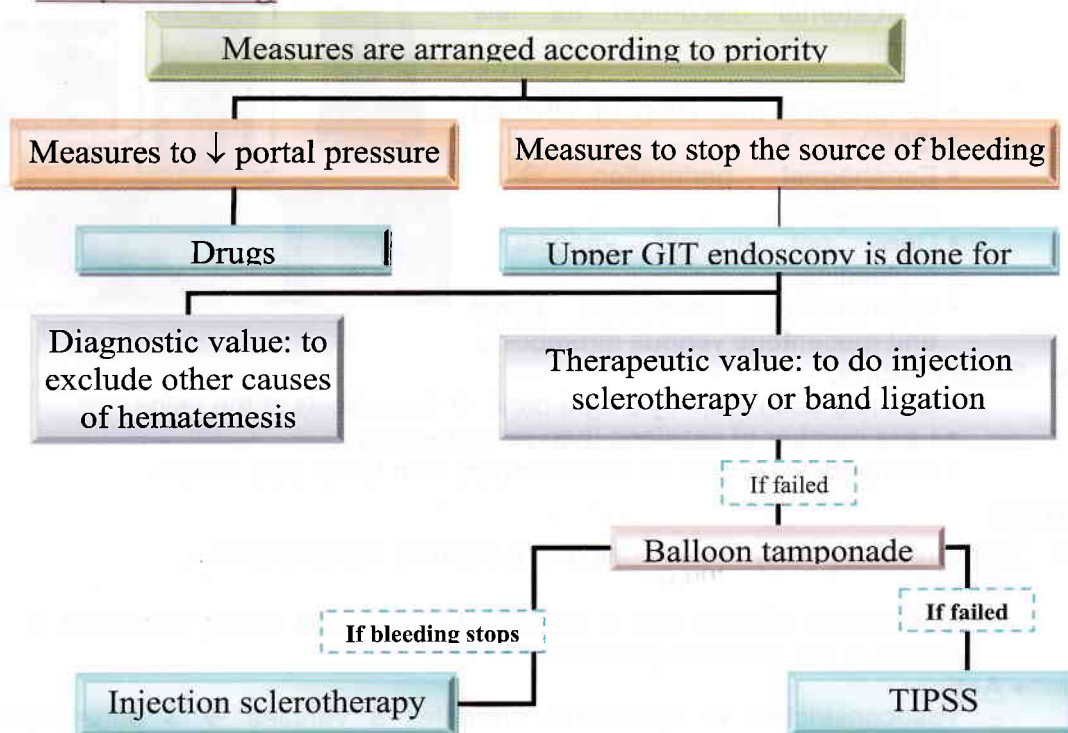
- IV line: IV fluids.
- Ryle.
- Catheter: to monitor urine output.
- Monitoring of: BP, pulse.
- Restoration of blood volume:
 - By fresh blood transfusion.
- Correct coagulopathy by:
 - Fresh blood (more platelet & coagulation factors & less ammonia & K).
 - Fresh frozen plasma.
 - IV vitamin K.
 - Hemostatics e.g. cyclokapron, Dicynon.
- Sedatives:
 - Avoid morphine and pethidine due to liver affection (we can use paraldehyde or chloral hydrate).

C- Prevent encephalopathy:

- **Aim:** evacuate blood from GIT & prevents its fermentation.
- **Method:**
 - Gastric lavage through Ryle tube by cold saline every hour.
 - Enema every 2 hrs.
 - Enteral antibiotics e.g. neomycin 1 gm / 6 hrs or Metronidazole 250 mg / tds
 - Lactulose (10 – 30 ml tds orally). It is fermented to lactic acid → combines with ammonia and has mild laxative effect.
 - Avoid hypokalemia and sedatives.

➤ **If encephalopathy is established, we add the following:**

- Flumazenil, branched aminoacids & L-dopa.
- Sedatives, if the patient is agitated.
- Care of the comatosed.
- Artificial biological liver.

D- Stop bleeding**i. Urgent upper GIT endoscopy:**

- Confirm the source of bleeding (**diagnostic**).
- For injection Sclerotherapy or band ligation (**therapeutic**).

➤ Injection Sclerotherapy

- Most effective treatment that preserve hepatic portal perfusion.
- Must be done as early as possible once the patient has been resuscitated.
- Done under mild sedatives as diazepam.

• Method:**▶ Intravariceal:**

- Injection into each varix to produce thrombosis:
 - 5 ml ethanolamine oleate (in esophageal varices).
 - Histoacryl blue "Bucrylate" (in gastric varices).

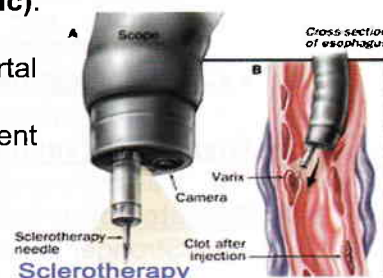
▶ Paravariceal: sub-mucosal injection of sclerosant.

- 1/2 ml of aethoxysclerol is injected to produce perivascular fibrosis.

- Both methods may be combined.
- Injection must be repeated until the varices are obliterated.

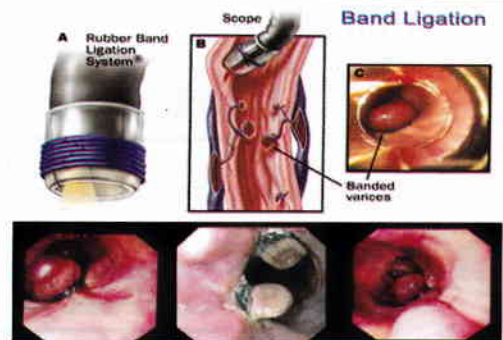
• Result:

- Stops bleeding in 90% of cases.



- **Side effects:**

- Retrosternal discomfort for few days.
- Fever.
- Esophageal ulceration or stricture (25%).
- Esophageal perforation → mediastinitis.
- Does not prevent variceal rebleeding.
- Sclerotherapy associated portal and mesenteric venous thrombosis



- **Band ligation:**

- Encircle each varix by a tight band → thrombosis of the veins.
- Less number of sessions than sclerotherapy.
- Comparable results as sclerotherapy with fewer side effects.

ii. Drugs:

- **Vasopressin** (the synthetic analogue(8-arginine vasopressin))

- **Dose:**

- Continuous infusion into a peripheral vein at 0.2 unit/kg dissolved in 200ml of 5% dextrose given over 20 min.

- **Action:**

- vasoconstriction of splanchnic arterioles & venules → ↓ portal venous pressure (portal vein has no smooth muscle)
- Also capable of constricting esophageal smooth muscles.

- **Side effects:**

- Colicky abdominal pain, diarrhea.
- Cardiac and cutaneous ischemia (can be reduced by simultaneous administration of nitroglycerine)

- **Contraindication:**

- Elderly patients and cardiac patients.

- **Glypressin (vasopressin + Glycine):**

- Has more prolonged action & fewer side effects.

- **Somatostatin:** (tetradecapeptide) given intravenously.

- Stronger action and fewer side effects
- Very expensive.

- **Octreotide:** longer acting analogue of somatostatin.

- **Propranolol:**

- Lowers portal pressure, reduce liver blood flow and cardiac output.
- Prevent recurrent variceal bleeding in compensated cirrhotic patients.

iii. Balloon tamponade: by:

Types

1. **Linton-Nachlas tube:**

- Has a large gastric but no esophageal balloon.

2. **Sengstaken-Blakmore tube:** (3 lumen)

- Have both gastric & esophageal balloons.
- Effective in cases of gastro-esophageal bleeding.

3. Minnesota: (4 lumen tube).

The fourth lumen is for aspiration of saliva from esophagus

- The gastric balloon is inflated by 200 ml air and pulled upward to press gastric fundus.
- If bleeding continues, the esophageal tube is inflated and the pressure in it should not exceed 40 mm Hg.
- This therapy is effective to stop bleeding in 80 – 90 % of cases
- **Disadvantages**
 - Discomfort sensation.
 - The patient can not swallow his saliva.
 - Esophageal rupture or pressure necrosis[☐].
 - Rebleeding in 60 – 80 % of patients[☐]
 - Rupture of gastric balloon → suffocation and death.
- To avoid these disadvantages patient should be in ICU for monitoring and insertion of endo-tracheal tube to prevent aspiration pneumonia.

iv. Transjugular intrahepatic porto-systemic shunt (TIPSS):

- It is the recommended method if all the previous methods failed to stop bleeding.
- It has proved very effective in the control of variceal hemorrhage.
- **Complications:**
 - 1- Perforation of the liver capsule leading to fatal hemorrhage.
 - 2- Porto-systemic encephalopathy in 40% of patients.
 - 3- Shunt occlusion 50% after one year.
- The results of shunt operations and esophageal stapling in patients with bleeding varices are not satisfactory and have a high mortality rate.

2-Silent Varices

- Prevention of bleeding from silent varices is important since the lifetime risk of a first-time variceal bleed in a cirrhotic patient is 30% and carries a mortality rate of up to 50%.
- So endoscopic screening should be done in all cirrhotics.
- Primary prevention with a non-selective beta-blocker (propranolol or nadolol) is started if large varices are found, especially if they have stigmata of bleeding (red streaks on the variceal surface).
- Primary prevention decreases the risk of a 1st variceal bleed by up to 40%.

3- Patients with Past History of Bleeding Esophageal Varices

According to Child Pugh classification[☐]

	1 point	2 points	3 points
Serum Bilirubin	0-2 mg %	2-3 mg% [☐]	≥ 3 mg%
Serum albumin	> 35 g/l	30-35 g/l	< 30 g/l
Ascites	None	Easily Controlled	Poorly Controlled
Encephalopathy	None	Mild or moderate	Advanced
Nutrition	Excellent	Good	Poor
A:	5-7 point	Suitable for surgery	
B:	8-11 point	Marginally suitable for surgery	
C:	12- 15 point	Unsuitable for surgery	

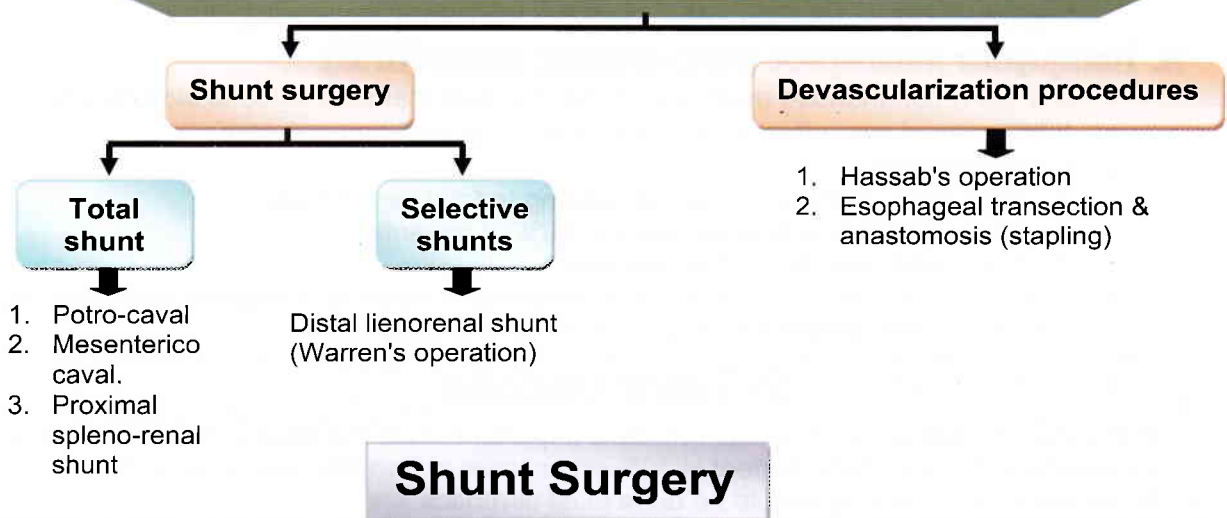
Recently:

- Whatever the category, repeated sclerotherapy is indicated until the varices are obliterated.
- **If Sclerotherapy fail:**
 - Child A: Porto-systemic shunt or devascularization.
 - Child B: Devascularization only.
 - Child C: Not fit for surgery, the only hope is liver transplantation.

Rule of 2/3 of portal hypertension

- 2/3 of patients with liver cirrhosis → will develop portal hypertension.
- 2/3 of patients with portal hypertension → will develop esophageal varices.
- 2/3 of patients with esophageal varices → bleed from varices.

Surgical treatment of portal hypertension



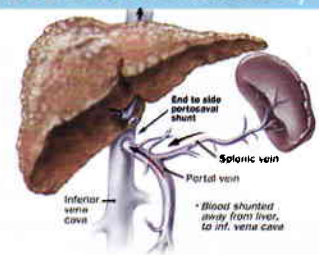
A. Total Shunt (shunt whole portal blood into the systemic circulation)

1- Porto-caval Shunt:

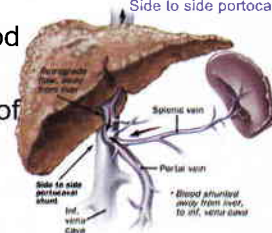
- The distal end of the portal vein is divided and transected while the proximal end is anastomosed to the anterior surface of IVC.
- The operation is very efficient in lowering the portal pressure and so no further bleeding occurs from the varices.

➤ Disadvantages:

- The operation deprives the liver from the portal blood flow, which accelerates the onset of liver failure.
- Recurrent hepatic encephalopathy in 30-50% of patients.



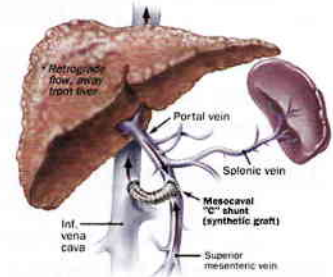
End to side portocaval shunt
Side to side portocaval shunt



2- Meso-caval shunt (Drapanas):

- **Idea:**
 - Insert a jump graft between SMV & IVC.
 - Which is either:
 - Wide bore prosthetic material Or
 - Autogenous saphenous vein.
- **Complications:**
 - Risk of encephalopathy is less than porto-caval shunt.
 - The incidence of thrombosis is high.

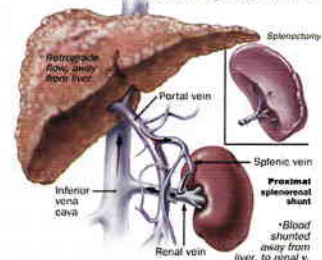
Mesocaval "C" Shunt



3- Proximal spleno-renal shunt:

- **Indications:**
 - Portal vein thrombosis.
 - If splenectomy is indicated
- **Technique:**
 - Splenectomy is performed then the proximal end of splenic vein is anastomosed to Lt. renal vein.

Proximal Splenorenal Shunt

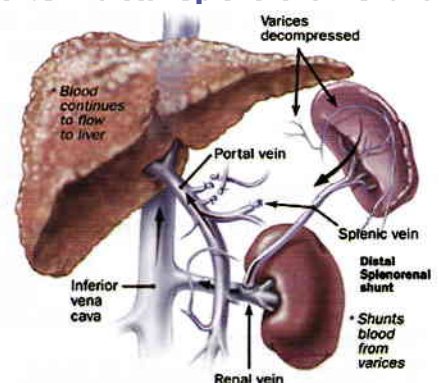


B. Selective Shunt

1. Distal lienorenal shunt (Warren's operation):

- **Idea:**
 - Selective decompression of gastro-esophageal varices without disturbing mesenteric or portal venous flow.
- **Technique:**
 - The Rt. & Lt. gastric vessels are ligated.
 - The splenic vein is mobilized; its proximal end is ligated, while the distal end is anastomosed to the Lt. renal vein.
 - The short gastric veins are preserved and will decompress the lower end of esophagus.
- **Advantages:**
 - **Risk of encephalopathy:**
 - Theoretically this should ↓ risk of porto-systemic encephalopathy (lowest risk).
 - **Portal venous flow to liver:**
 - Maintained → LF not impaired.

Warren "Distal Splenorenal" Shunt



➤ **Disadvantages:**

- ↑Incidence of thrombosis.
- Pancreatic siphon (after few years it turns into total shunt due to presence of connections into the pancreas that connects the splenic & superior mesenteric veins).
- Porto-systemic encephalopathy (shifting of blood from high to low pressure with ↑ risk of encephalopathy).

Devascularization Procedures

(Porto azygos disconnection)

- They are non-shunting procedures used to treat gastro-esophageal hemorrhage.
- Best treatment of variceal bleeding 2ry to portal vein splenic & S.M.V. thrombosis ☐.

➤ Indications of devascularization operations:

1. Elective cases:-
2. Failure of sclerotherapy in patients with good general condition.

1. Hassab-Khairy Operation ☐:

➤ Advantages:

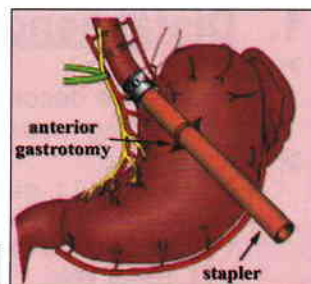
- No encephalopathy.
- Portal flow is intact.
- Low incidence of rebleeding.

➤ Trans-abdominal:

1. Splenectomy.
2. Devascularization of lower 5-10 cm of the esophagus.
3. Devascularization of proximal part of the stomach (right and left gastric vessels and short gastric vessels leaving only right gastroepiploic vessels).
4. Vagotomy + pyloroplasty (patients liable for PU).

2. Esophageal Transection & Anastomosis:

- The purpose of this operation is interruption of submucosal venous plexuses which is still intact following porto azygos disconnection.
- It is simple and easy and can be added to porto azygos disconnection.



Liver transplantation

- Is indicated for patients with end-stage liver disease.
- It will correct the problem of the varices and ascites.

Management of ascites

1. Rest in bed:

2. Restriction of salt & fluid:

- Follow up by → urine volume, fluid chart, body weight.

3. Diuretics:

- Spironolactone.
- Frusemide + Aldactone.

4. Salt free albumin:

- ↑ Osmotic pressure of plasma.

5. Tapping: If there is difficulty in respiration.

6. Resistant ascites:

- Not responding to salt and fluid restriction & lasix and aldosterone for at least 2 weeks.

➤ Causes:

- a) Salt retention.
- b) Marked hypoalbuminemia → ↑ albumin.
- c) Impaired kidney function.
- d) Malignant ascites e.g. hepatoma.



➤ **Treatment:**

- a) Denver shunt[☐] (between internal jugular vein & peritoneum) DIC is the most feared complication[☐].
- b) Infusion of salt poor albumin[☐].
- c) Ultrafiltration & reinfusion.
- d) TIPSS[☐].
- e) Liver transplantation.

Surgical key points on Ascites and Portal Hypertension**1) Spontaneous bacterial peritonitis:**

- See peritoneum

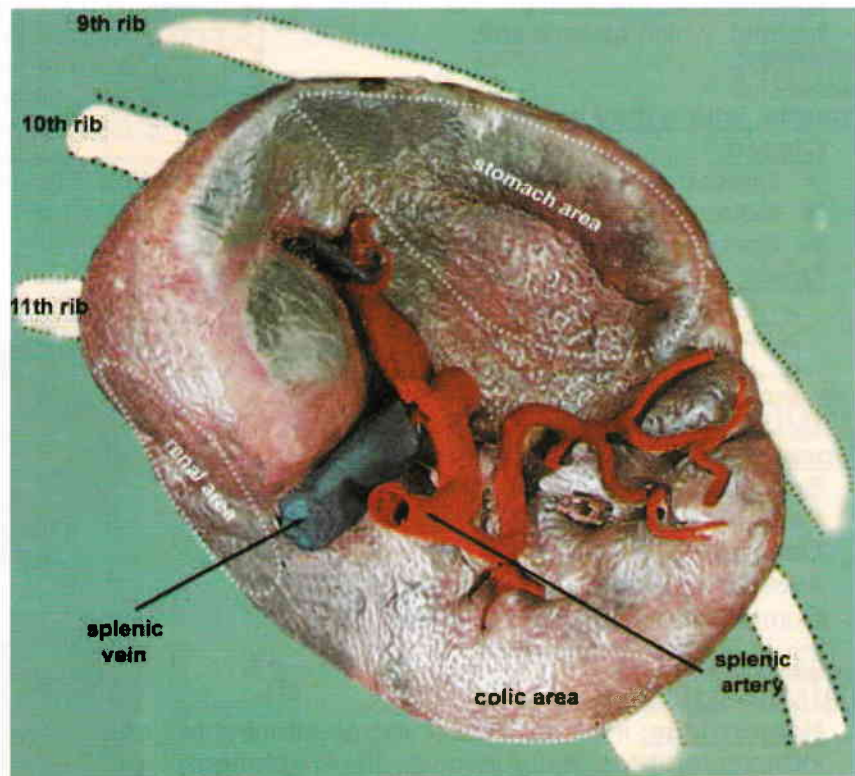
2) Therapeutic paracentesis:

- Limited for patients with tense or symptomatic ascites.

3) Peritoneo-venous shunts:

- **Idea:** they result in unidirectional flow of ascitic fluid from peritoneal cavity into venous circulation.
- **Indication:** disabling ascites refractory to conventional medical treatment and therapeutic paracentesis.

CHAPETR 10



THE SPLEEN

Rupture of the Spleen

- It is the most common organ to be injured in blunt abdominal trauma[☑].

Predisposing Factors

- Splenic enlargement: which makes it more liable to trauma.
- Diseases of the spleen: e.g. malaria & typhoid, which make it soft.

Etiology

➤ Trauma, which may be:

- **Closed:**
 - Direct trauma (blunt trauma) e.g. motor car accident & falling from a height.
 - Indirect trauma: fracture ribs.
 - Spontaneous rupture: with pathological spleen.
- **Open:**
 - Gun-shot wounds.
 - Puncture due to stabbing.
 - Iatrogenic: e.g. gastrectomy.

- ▶ Commonest abdominal organ to be injured
- ▶ **Etiology:** Trauma (closed or open)
- ▶ **C/P:** History of trauma, shock, signs of blood in peritoneum (acute abdomen)
- ▶ **Invest.:** U/S & CT (most useful), DPL
- ▶ **TTT:** a. ABCD + preoperative preparation
b. Adult → immediate laparotomy & splenectomy
c. Child → splenic preservation

Pathology

➤ Types of ruptured spleen:

- Subcapsular hematoma.
- Superficial tear(s).
- Deep tear(s).
- Avulsion of a pole of the spleen.
- Complete depulping of the spleen.
- Injury of a vascular pedicle.

Complications

- **Hemorrhage:** internal (intra or extraperitoneal or intrapleural if the injury involves the diaphragm) & external.
- **Splenic cyst:** may follow a perisplenic hematoma.
- **Associated** abdominal or thoracic **injuries**.

Clinical Picture

History

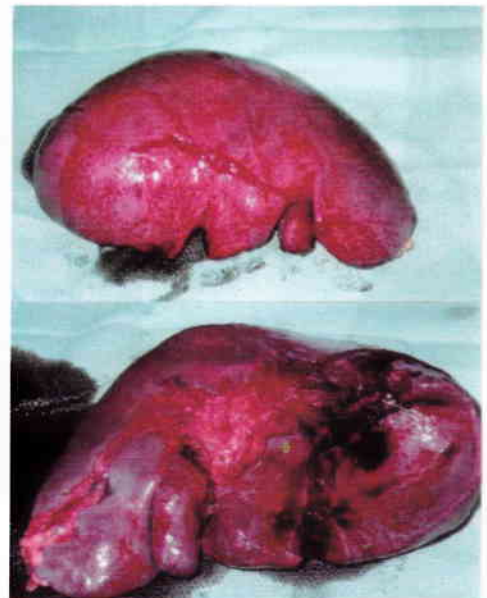
- History of trauma to the upper abdomen or lower chest followed by abdominal pain.

Examination

There are three types:

1- Classic type (commonest presentation):

- Three stages will be present:
- **Stage of shock** → **stage of recovery** from shock (Lucid interval) due to arrest of bleeding from hypotension → **stage of internal hemorrhage** due to rise of blood pressure after resuscitation.



Splenic lacerations

⇒ **General examination:**

- Rapid weak pulse, hypotension.
- Subnormal temperature, cold extremities & pallor.
- Decrease in urine output despite adequate fluid replacement.

⇒ **Local examination:**

- Inspection:
 - Bruises in the Lt. Hypochondrium.
 - Fracture of the Lt. Lower ribs.
- Palpation:
 - Tenderness & rigidity in the Lt. hypochondrium (could be minimal).
 - Rebound tenderness.
- Percussion: shifting dullness.
- Auscultation: ↓ intestinal sounds.
- DRE: tenderness, fullness in the rectovesical pouch.

⇒ **Special signs**☑:

- **Kehr's sign:** referred pain to the Lt. Shoulder ± hyperesthesia from diaphragmatic irritation by blood, especially with Trendelenberg position.
- **Cullen's sign** (late): bluish discoloration around the umbilicus due to absorption of the intraperitoneal blood by lymphatics around the umbilicus.
- **Balance's sign** (late & pathognomonic):(+ve in 25%of cases)
 - Shifting dullness on the Rt. side from the free blood in the peritoneum.
 - Fixed dullness on the Lt. side due to clots & retroperitoneal hematoma.

- Despite these traditional signs, continued blood loss from bleeding vessels on the surface of the torn spleen is hard to diagnose.

2- Fatal type:

- Due to tearing of the splenic vessels or complete avulsion of the splenic pedicle.
- The patient is severely shocked & usually dies within a short period.
- Usually associated with other visceral injuries.

3- Delayed type:

- The initial shock is followed by long Lucid interval up to 15 days, after which the patient presents with features of internal hemorrhage. This delay is due to:
 - Formation of subcapsular hematoma which may be ruptured later.
 - The greater omentum seals the splenic tear then retracts later on.
 - Blood clots seal the splenic vessels then dislodges when BP rises.

Investigations

▪ **The most important diagnostic tools are**☑:

- Abdominal U/S & CT scan.

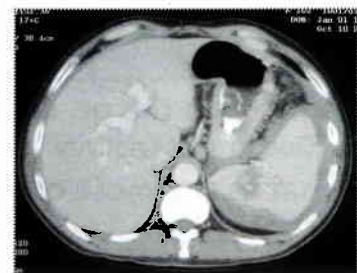
I. Radiological:

▪ **Abdominal U/S & CT scan (most useful):**

- Are diagnostic & show pathological type of rupture.
- Shows surrounding hematoma.
- Serial examination showing change in splenic size can identify enlarging sub-capsular hematoma.
- Free blood in the peritoneal cavity.

▪ **Plain x-ray:**

- Fracture in the lower left ribs.
- Elevated left copula of the diaphragm☑.
- Loss of splenic outlines.
- Indentation of fundic air bubble☑.
- Obliteration of left psoas shadow☑.



Splenic lacerations

2. Instrumental:

- **Diagnostic peritoneal lavage** [☑] (**greatly replaced by U/S and CT**):
- Intra-peritoneal hemorrhage is diagnosed if there is > 100,000 RBCs/ml.

3. Laboratory:

- CBC, KFTs, FBS, electrolytes ...

If the patient is in severe shock there is no time for investigations and the surgeon should depend on clinical findings for surgery.

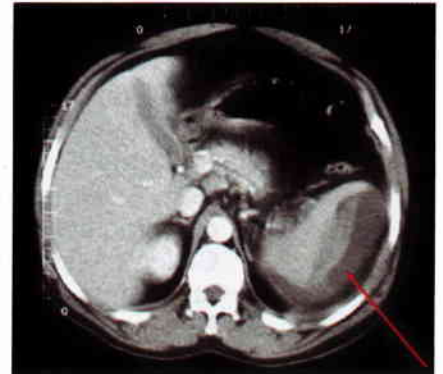
Treatment

Pre-operative Preparation

- ⇒ Blood transfusion, morphia.

Surgery

- ⇒ Adult → immediate laparotomy & Splenectomy.
- ⇒ Children → **splenic preservation** [☑] by:
 - Splenorrhaphy: suturing of small tears.
 - Splenic artery ligation (occasionally indicated with splenorrhaphy)
 - Partial Splenectomy: if avulsed one pole.
 - Splenic embolization by gel foam.
 - Splenic mesh wrap: placing the injured spleen in the centre of the mesh & sewing both ends of the mesh together to tamponade the bleeding.
- **Precautions should be done if Splenectomy is indicated before five years old:**
 - **Preoperative:** vaccination against the capsulated organisms [☑] (pneumococci, meningococci, H. influenza). Pneumococci is the commonest [☑] cause of OPSI*.
 - **Pneumovax** is given every 5 years until the age of 18.
 - **Postoperative:** long acting penicillin.



Traumatic subcapsular hematoma

*OPSI = overwhelming post splenectomy infection.

➔ Policy of conservative treatment after blunt abdominal trauma:

1. Minimal injury in (liver, spleen, kidney).
 2. Good general condition.
 3. Bleeding is not progressive, while in penetrating injury we have to explore especially if penetrating intestine.
- Resuscitation, rest in bed & meticulous follow up for up to 7 – 10 days.

Q: Why does the pt. complain of bone ache after splenectomy?

- ⇒ Due to ↑ BM activity.

Q: what is the most common complication of splenectomy?

- ⇒ Hemorrhage.

Causes of Splenomegaly

1. Infective causes:

- Bacterial (Typhoid & paratyphoid, septicemia, pyogenic abscess, tuberculosis, typhus).
- Viral (infective hepatitis, infectious mononucleosis).
- Spirochaetal (Weirs disease, syphilis)
- Parasitic (Bilharziasis, hydatid cyst, malaria, trypanosomiasis, kala azar).

2. Blood diseases:

- Leukaemias.
- Haemolytic anaemias (Spherocytosis, Cooley's anaemia, acquired haemolytic anaemia, hypersplenism).
- Pernicious anaemia.
- Polycythaemia rubra vera.
- Idiopathic thrombocytopenic purpura.

3. Congestive splenomegaly:

- Due to portal hypertension.

4. Metabolic:

- Gaucher's disease, amyloidosis, porphyria, rickets.

5. Tumors:

- Hemangioma, fibrosarcoma, lymphomas (commonest cause of neoplastic enlargement of spleen[□]).

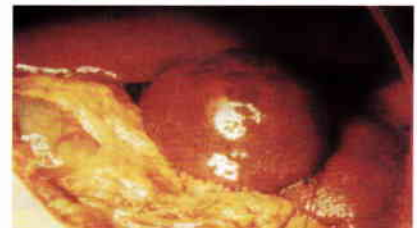
6. Non parasitic cysts.

7. Collagen diseases:

- Felty's syndrome and Still's disease.



Splenomegaly in Hairy Cell Leukemia



Splenic Cyst

Q: What is meant by huge splenomegaly?

Enlarged spleen crossing the umbilicus.

Q: What are the causes of huge Splenomegaly?

Bilharzias, chronic myeloid leukemia- chronic malaria- Kala Azar, thalassemia major, myelofibrosis, amyloidosis, Gaucher's disease

Bilharzial splenomegaly:**✿ Causes of splenomegaly in bilharziasis:**

- 1- Hyperplasia of RES due to absorption of Bilharzial toxins.
- 2- Bilharziasis → periportal fibrosis → portal hypertension → congestive Splenomegaly.

✿ Pathology:**➤ Macroscopic Picture:**

1. **Capsule** → thickened with adhesions 2ry to perisplenitis (due to subcapsular hemorrhages & thrombosis in the blood vessels followed by some infection).
2. **Cut surface** → deeply congested + prominent fibrous trabeculae.
3. **The vessels in the pedicle** → large, dilated & tortuous
4. **The ligament** → elongated & narrowed.
5. **Prominent notch.**

✿ Treatment:

- Splenectomy in patients with Egyptian hepatosplenomegaly is indicated only in the following conditions:
 - 1- Hypersplenism.
 - 2- Huge splenomegaly.
 - 3- Recurrent attacks of pain 2ry to splenic infarctions.
 - 4- As a part of operations done for portal hypertension.

Types of anemia improved after splenectomy: ✓

- 1- Spherocytosis.
- 2- Thalassemia.
- 3- Hypersplenism associated anemia.

Splenomegaly + lymphadenopathy ✓

- 1- Hodgkin's disease.
- 2- Acute leukemia.
- 3- Felty's syndrome.

Indications for elective splenectomy: ✓

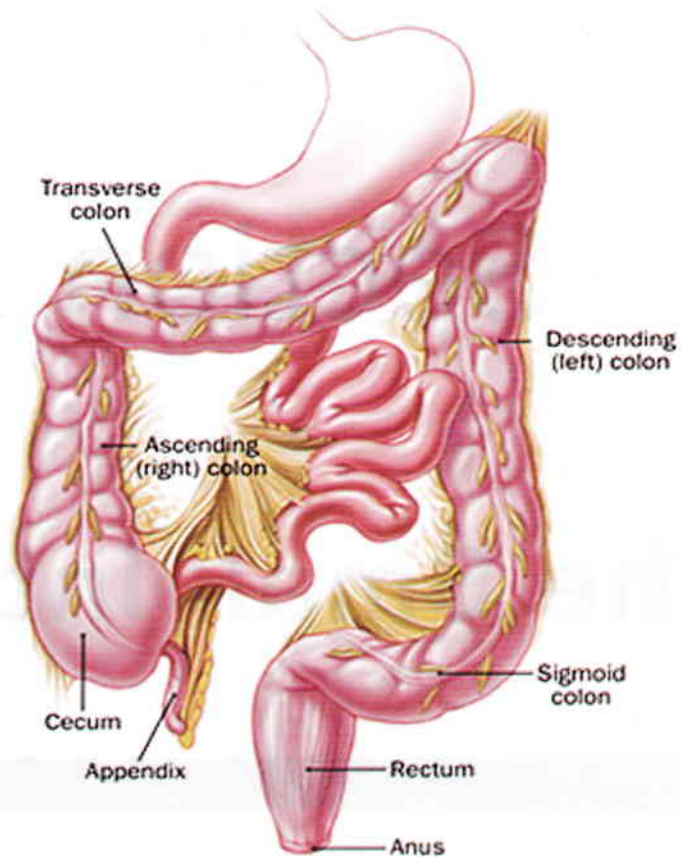
- 1- Hypersplenism.
- 2- Staging of Hodgkin's disease.
- 3- To treat splenic cyst, tumor or abscess.
- 4- Hemolytic anemias except sickle cell anemia. ✓✓

Purpura is due to reduced platelets, thrombocytopathies, and increased capillary fragility ✓.

Petechiae: is due to rupture capillaries.

Thrombocytopenia can be due to aplastic anemia, DIC and hypersplenism ✓, and not caused post-splenectomy.

CHAPETR 11



THE INTESTINE

Section A

The Small Intestine

Meckel's Diverticulum

Definition of Diverticulum

- A blind pouch that is continuous with the lumen of a hollow viscus (gut or urinary bladder).
- **Meckel's Diverticulum**
- It is the most common cause of bleeding per rectum in childhood

Definition of Meckel's Diverticulum

- Persistent patency of the proximal part of the vitellointestinal duct.

Incidence (Rule of 2)

(Most prevalent congenital anomaly of the GIT)

- 2% of people are affected.
- 2% of affected people may have complications
- 2 times more common in males than females.
- 2 feet (60 cm) from ileocaecal junction.
- 2 inches long.

Pathology

- It is a true diverticulum → consists of all layers of bowel wall & has a separate blood supply from SMA.
- It projects from antimesenteric borders.
- It may contain ectopic tissues: gastric, pancreatic, duodenal or colonic (20% of cases).

Clinical Picture

- Asymptomatic.
- C/P of complications (see below).
- The most frequent manifestation is bleeding.

Complications

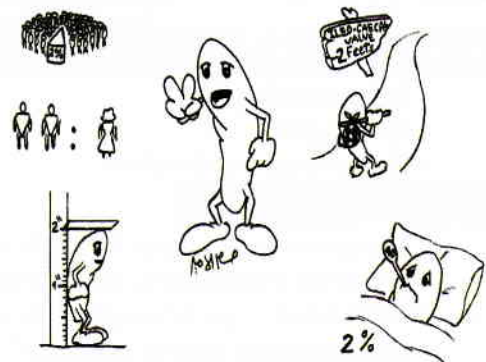
1. Intestinal Obstruction (COMMONEST):

- It may be due to:
 - **Intussusception:** the ectopic mucosa at the base of the diverticulum acts as FB and forms the apex of an ileoileal intussusception.
 - **Volvulus:** due to pressure on an intestinal loop by a fibrous band that may attach the diverticulum to the umbilicus. The band may also allow rotation of the ileum around its axis.

2. Peptic ulceration:

- Sometimes the diverticulum contains ectopic gastric epithelium.
- This leads to formation of peptic ulcer on the neighboring epithelium.
- **The ulcer is liable to:**
 - Perforation.
 - Bleeding → melena or BPR according to the amount of bleeding.
- **Clinical Picture:**
 - Usually a child 8-10 years old.
 - Abdominal pain (around the umbilicus because it is a part of Midgut) following meals
 - Melena.
 - Picture of acute peritonitis if the ulcer perforates.

- ▶ It is a true diverticulum.
- ▶ **C/P:** Asymptomatic + C/P of complications
It is the commonest cause of bleeding per rectum in **children**
- ▶ **Comp.:** IO, p. ulcer, diverticulitis, Littre's h.
- ▶ **Invest.:** a- Barium meal follow through
b- Angiography
- ▶ **TTT:** a. **Symptomatic:** excision of the diverticulum
b. **Silent cases:** leave it alone unless excision is indicated



Rule of 2 in Meckel's

3. Meckel's Diverticulitis:

➤ diagnosis:

- Clinical picture similar to that of acute appendicitis.
- At operation, the appendix is found normal (it is the only way to differentiate between acute appendicitis and Meckel's diverticulitis).

➤ It is more dangerous than acute appendicitis due to ☑:

- Easier perforation due to its thin wall.
- More difficult localization due to its central position & mobility of the small intestine.

Perforated Meckel's diverticulum should be differentiated from:

- Perforated DU.
- Perforated appendix.



4. Littre's Hernia

- May strangulate causing strangulation without obstruction.

DD of bleeding per rectum in a child:

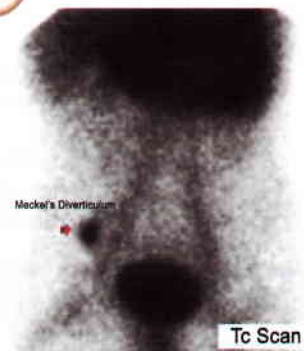
- 1- Meckel's with PU.
- 2- Angiomatous malformation.
- 3- Typhoid ulcers.
- 4- Colonic polyp.
- 5- Piles (rare).

Differential diagnosis ☑

- Appendicitis.
- Salpingitis.
- Perforated peptic ulcer.

Investigations

- Barium meals follow through: may show a diverticulum.
- Superior mesenteric angiography: extravasation.
- Tc⁹⁹ Scan + gamma camera: safer than arteriography can demonstrate gastric mucosa ☑.
- Angiography → extravasation: **weeping meckel's** (active blood, 0.5 ml / min)



Treatment

▪ Symptomatic cases:

- Wedge resection of the ileum and closure of the defect transversely or segmental resection is indicated.

▪ Silent cases and accidentally discovered at laparotomy:

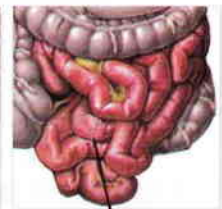
- Indications of resection ☑ (if complications are likely):

- 1- Narrow mouthed (diverticulitis is likely).
- 2- The wall of the diverticulum is thickened (contains ectopic gastric tissue)... (PU is likely).
- 3- Attached band of adhesions... (I.O. is likely)
- 4- Young adults and children... (Different complications are likely).

- If not → we can leave it ☑.



Meckle's diverticulum



excision of Meckle's diverticulum

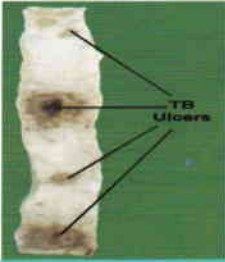
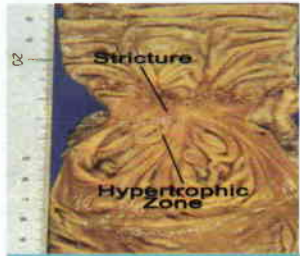
➤ Intestinal Diverticulae:

1. Diverticular disease of small intestine.
2. Diverticular disease of the colon.

- Most common cause of bleeding per rectum in childhood is Meckel's diverticulum.

Inflammatory Diseases of the Small Intestine

1- TB of the Intestine

	Ulcerative type	Hypertrophic type (<10%)
1. Etiology: a. Organism b. Route c. Predisposing factors - Age - Immunity - Virulence	▪ Mycobacterium tuberculosis. ▪ 2nd to pulmonary TB. ▪ Swallowing of infected sputum. Adult Usually bad (open case of TB)	▪ Mycobacterium bovis. ▪ Ingestion of the TB bacilli. ▪ Ingestion of infected milk. Child relatively good least virulent
2. Pathology: (Lesion)	TB Ulcer: - Site → terminal ileum (more lymphatics) - Result → transverse ulcers (lymphatics circumferentially) - Number → multiple. - Edge → Undermined (affection of connective tissue > epithelium) - Margin → Cyanotic (due to EAO). - Floor → covered with caseous material. - Base → Indurated. - Serosa → may be studded with tubercles and thickened.  Ulcerative	- Site → ileocaecal region. - Result → thickening of submucous and subserosal layers → fibrosis → narrowing of terminal ileum, caecum, ascending colon. - Mesenteric LNs → involved early & may caseate. - No gross caseous necrosis.  Hypertrophic
3. Clinical Picture:	General - Manifestations of pulmonary TB - ↓ weight & anemia Local - Diarrhea, - Colicky lower abdominal pain - Foetid bloody stool.	General - ♀ > ♂ - ↓ weight & anemia Local - Diarrhea - Abdominal pain - Fixed firm tender mass in Rt. iliac fossa. - Sometimes recurrent episodes of subacute IO.

	Ulcerative type	Hypertrophic type (<10%)
4. Complications	- Stricture formation → IO. - Perforation (rare)	- Stricture → IO. - Fecal fistula
5. Investigations	Laboratory: - CBC anemia, lymphocytosis - ESR & +ve C reactive protein - +ve tuberculin test - Stool culture on Lowenstein Jensen media - PCR - Abdominal X-ray may show extensive calcification. Radiological: - Ba meal follow through [showing +ve sterlin sign (non visualization of the terminal ileum & caecum)]	Radiological: - Ba meal follow through [Showing narrowing of the ileum with elevated caecum]
6. Treatment	- Anti TB drugs + sanatorial ttt - Surgical ttt: <u>Indicated for:</u> perforation, stricture & bleeding. <u>Operation:</u> resection and anastomosis.	- Surgical ttt: <u>Indicated for:</u> perforation, fecal fistula & obstruction. <u>Operation:</u> right hemicolectomy



Barium meal follow through showing Sterlin's sign



Barium meal follow through showing Hypertrophic TB

DD

- Cancer of the caecum.
- Appendicular mass.
- Crohn's disease.
- Actinomycosis.

2- Surgical complications of Typhoid and Paratyphoid

➡ **COMPLICATIONS:**

○ **Intestinal complications:**

- **Paralytic ileus** (the commonest complication).
- **Intestinal Hemorrhage:**
 - Due to ulceration of the affected peyer's patches.
 - The ulcer is parallel to the long axis of the gut.
 - Bleeding usually occurs 2-3 weeks after the onset of the disease.
 - It is usually dark blood per rectum but may be bright red (in 10-20% of hospitalized patients).
- **Perforation of the typhoid ulcer (2%):** may occur around the 3rd week with resulting peritonitis.

○ **Biliary complications:**

- Acute non-calicular cholecystitis.
- Chronic cholecystitis.

○ **Vascular complications:**

- DVT of the lower limbs.

○ **Musculoskeletal complications:**

- Zenker's degeneration of rectus abdominus muscle.
- Arthritis and osteomyelitis.

Miscellaneous Small Intestinal Diseases

Small intestinal fistula

- Fistula is an abnormal communication between two epithelial surfaces.

Etiology

- Injury during operation (in majority of cases)
- **C**rohn's disease.
- **C**olonic diverticulitis.
- **C**ancer.
- **R**adiation enteritis.
- **I**ntestinal tuberculosis.
- **M**esenteric vascular disease.

Types

1- Internal fistula : connects intestine to another hollow viscus

2- External fistula:

- High (>500 ml / day) and low(<500 ml / day) fistulas depending on their location and output.
- The more proximal the fistula the more the output, the more serious the problem.

Complications

- Sepsis (intraperitoneal abscess).
- Skin necrosis.
- Malnutrition and fluid and electrolyte abnormalities.

Investigations

- Barium meal follow through.
- Barium enema.
- Fistulogram.

Treatment

Conservative management

- **Nutritional support by I.V fluids**
- **Control of sepsis**
 - Sumb suction helps to control sepsis and provide drainage for associated intra-abdominal abscess.
 - Nasogastric tube in patient with ileus helps to reduce output from fistula.
 - Somatostatin also inhibits intestinal secretion and motility.
 - Systemic antibiotics should be given until sepsis is controlled.
- **Skin stoma care**
- **With modern conservative management, spontaneous closure occurs in 70-80% of cases and mortality averages 6-10%.**

Surgical treatment

A- External fistula :

➤ Indications :

1. If there is still significant output after 4-6 weeks of conservative management.
2. Distal obstruction.
3. Affected segment has active granulomatous disease.

B- Internal fistula:

➤ Spontaneous closure is rare

➤ Surgery for:

1. Fistula with urinary tract
2. If the fistula passes a long segment

Intestinal trauma

Etiology

1. Blunt abdominal trauma as in RTA.
2. Penetrating trauma as stabs or bullets.
3. Iatrogenic trauma.

Types of injury

- 1- Contusion and hematoma.
- 2- Rupture which may be complete or incomplete and single or multiple.
- 3- Tears of the mesentery or the mesenteric blood vessels.

Sequelae

1. Peritonitis due to escape of intestinal contents.
2. Internal hemorrhage → It is less of a problem than peritonitis.
3. Hypovolemic and septic shock.
4. Paralytic ileus.

Clinical picture

Symptoms

- 1- History of trauma.
- 2- Abdominal pain.

Signs

- **General:** Tachycardia, fever and hypotension.
- **Local:**
 1. Signs of injury in the abdominal wall.
 2. Tenderness and rebound tenderness.
 3. Shifting dullness.
 4. Distension due to ileus.

► **Etiology:** open, closed, iatrogenic (like any trauma)

► **C/P:** a- History of trauma.

b- Abdominal pain

b- Signs of shock.

c- Signs of fluid in peritoneum

► **Invest.:** • The diagnosis of bowel injury is mainly based on clinical findings

• X ray & U/S are helpful

► **TTT:** a. R&M

b. All intestinal injuries require laparotomy.

- Whenever there is doubt of peritonitis, it is better to keep the patient under observation in the hospital for 24 hours.

Investigations

➤ **The diagnosis of bowel injury is mainly based on clinical findings**

1- **Laboratory** : Leucocytosis and hemodilution.

2- **Radiological**:

- Plain x-ray of the abdomen may show;
 - Free air under the diaphragm.
 - Multiple fluid levels due to ileus.
 - Fractures.
- U/S or CT scan: may show hematoma or intraperitoneal collection.

3- **DPL**.

Treatment

➤ **All intestinal injuries require laparotomy.**

➤ **Priorities of multiple trauma management should be followed.**

A) Preoperative preparation:

- Anti-shock measures.
- Antibiotics.
- Tetanus toxoid.
- Insertion of NGT and urinary catheter.

B) Operative TTT:

1. **Midline abdominal incision.**

2. **Abdominal exploration and arrest bleeding.**

➤ **Small intestine and right colon injuries:**

- Tidy sharp injuries are sutured in 2 layers.
- Ragged injuries require trimming of the edges and are sutured.
- Intestinal resection is indicated for ischemic or gangrenous segments.

➤ **Transverse and left colon injuries:**

- Localized injuries are exteriorized, thus acting as colostomy.
- If the injured part can not be exteriorized, the tear is sutured and protected by proximal colostomy.
- In either cases the colostomy is closed after 3 weeks.
- In cases where resection is indicated, the proximal end of the colon is brought out as terminal colostomy. The distal end is either brought out as mucous fistula or closed (Hartmann's pouch).
- Anastomosis is then done after 3 weeks.

Ileostomy

➡ **See operative book.**

Section B

Intestinal Obstruction



General considerations

Definition

- Arrest of the downward propulsion of the intestinal contents.

I- According to the Pathological Nature of the Cause:

1- Simple obstruction:

- Due to mechanical occlusion of the gut lumen without interference with its blood supply.

Pressure on the wall compresses also its blood vessels → predisposing to strangulation.

2- Strangulation and obstruction:

- There is significant interference of blood supply of the involved bowel segment, if not relieved within 6 hours → gangrene.

3- Paralytic ileus:

- Due to loss of propulsive power of the bowel leading to functional obstruction.

II- According to the Level of Obstruction

- High small bowel obstruction.
- Low small bowel obstruction.
- Large Bowel obstruction.

III- According to the Onset and the Course of Obstruction:

- Acute obstruction: (e.g. intussusception, MVO) → **especially in small intestine.**
 - Clinical course is rapid.
- Chronic obstruction: (e.g. Cancer colon) → **especially in large intestine.**
 - Clinical course is slowly progressive.
- Acute on top of chronic obstruction: → **sudden change in behaviour.**
 - When a narrowed lumen becomes totally occluded by dry stools.
 - e.g. - Cancer caecum with erosion of terminal ileum.
 - Cancer caecum with obstruction by fecolith.

IV- According to Motility:

- Dynamic obstruction: Due to mechanical occlusion of the lumen.
- Adynamic obstruction: due to:
 - Paralytic ileus (absent peristalsis)
 - Mesenteric vascular occlusion (non propulsive peristalsis)

Acute Intestinal Obstruction (General Principles)

Causes

1- Simple:

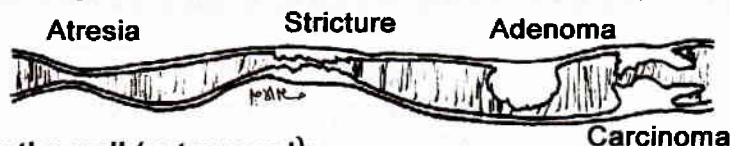
➤ In the lumen (intraluminal):

- e.g. faecal impaction, gall stone ileus and parasitic infestation.

- ▶ **Class.:** pathology, level, onset, motility
- ▶ **Etiology:** strangulated hernia, intussusception
- ▶ **C/P:** pain, distension, vomiting, absolute Constipation, may be visible peristalsis
- ▶ **Comp.:** simple, strangulated
- ▶ **Invest.:** X-ray erect, U/S, comp.
- ▶ **TTT:** pre-op. preparation, conservative, surgery

➤ **In the wall (intramural):**

- e.g. congenital atresia, tumors, chronic diverticulitis, Crohn's disease.



➤ **Outside the wall (extramural):**

- e.g. adhesions, strangulated hernia and volvulus.

2- **Strangulation:**

- Strangulated hernia, volvulus, intussusception & MVO.

Common Causes According to Age

Strangulated hernia is a common cause in different age groups.

1- **Neonates:**

- Congenital atresia, volvulus neonatorum, anorectal malformation, meconium ileus and Hirschsprung's disease.

2- **Infants:**

- Ileocecal intussusception, Hirschsprung's disease and strangulated hernia (commonest cause).

3- **Adults:**

- Post surgical adhesions (commonest cause).
- Strangulated hernia

4- **Elderly:**

- Strangulated hernia
- Cancer colon
- Sigmoid volvulus.
- Diverticular disease.
- Pseudo obstruction.(Ogilvie syndrome)



**Incarcerated faeces
causing Intestinal
Obstruction**

Pathology

1. **Simple obstruction:**

a- **Distal to the obstruction:**

- The bowel has normal peristalsis and absorption until it becomes empty then immobile.

b- **Proximal to the obstruction:**

I- The intestine becomes distended by:

➤ **Gases**☑:

- Swallowed air (68%): Patients with IO may swallow excessive amounts of air due to acute pain and anxiety.
- Diffusion from congested vessels (22%).
- Bacterial fermentation (10%).

➤ **GIT secretions:** 8 liters /day.

II- Hyperperistaltic waves:

- Sudden intestinal obstruction → stretch of the muscles of the proximal segment → hyperperistalsis (↑ power and frequency of contractions) in attempt to overcome the obstruction.
- If obstruction is not relieved → bowel dilates → ↓ peristalsis → more dilatation → bowel becomes flaccid and paralyzed (secondary ileus).

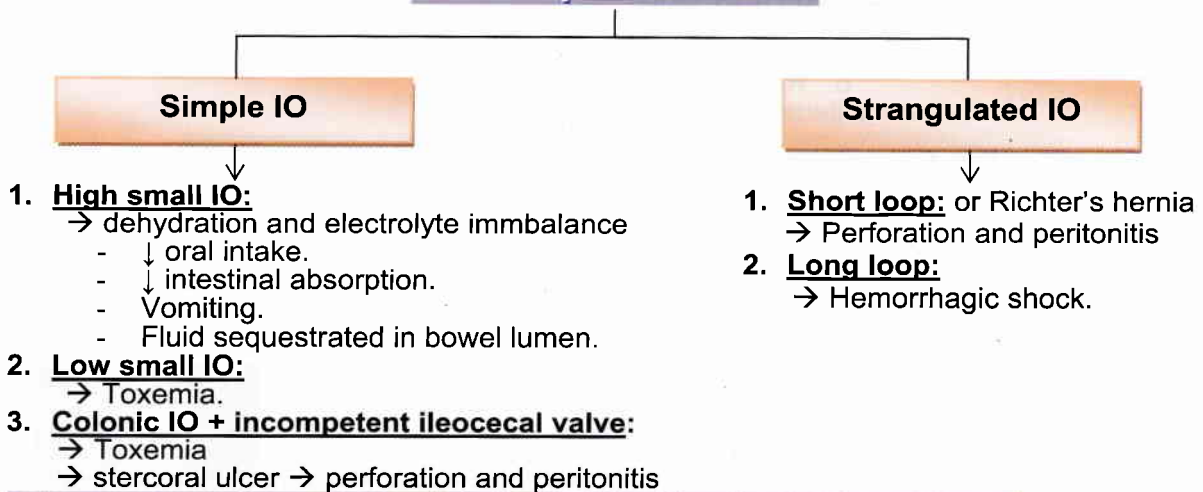
III- Ulceration and perforation due to either:

- More distension → more ischemia
 - More stasis → more infection
- } Ulceration & perforation → generalized peritonitis

2. Strangulation obstruction:

- In addition to the above
- Bacteria and toxins in the lumen can through ischemic bowel wall to the peritoneal cavity.
- Unrelieved strangulation can lead to septic shock.
- The mucosa is the 1st layer to be affected producing ulceration and intramural bleeding

Complications



Clinical Picture of simple intestinal obstruction

Symptoms {Pain, Distention, Vomiting, Absolute constipation[☑]}

- 1- Pain: (due to hyperactive peristalsis) usually the 1st symptom
 - Onset: -in high small IO → within a day
-In low small IO → within 2-3 days
 - Character: colicky.
 - Site:
 - In high small IO → above the umbilicus.
 - In low small IO → around and below the umbilicus.
 - In large IO → lower & peripheral parts of the abdomen.

- Painless → if paralytic ileus.
 - Severe stabbing pain → in case of ischemia.
- 2- Distension:
 - Marked in: colonic obstruction "flanks".
 - Mild or absent in: high small IO.
 - Central abdominal in: low small IO.
- 3- Vomiting: (caused by anti-peristaltic wave)
 - Onset:
 - High small IO → early with the onset of pain.
 - Low small IO → after few hours from the onset of pain.
 - Colonic obstruction → it may be delayed for 1-2 days.

- Content:
 - First: gastric juice (whitish mucoid).
 - Then: bile stained (greenish).
 - Finally: feculent (brown and offensive).

Vomiting may be absent in paralytic ileus ✓.

4- **Absolute Constipation:**

- Definition: No faeces or flatus is passed **inspite of desire**.
- Onset: Early in colonic obstruction, late in high small IO.
- May be absent in ✓:
 - 1- Early (due to stool distal to the obstruction).
 - 2- Intussusception (jelly-stool).
 - 3- Strangulation without obstruction:
 - a. Mesenteric vascular occlusion (bleeding per rectum).
 - b. Richter's or Littre's hernia.
 4. Gall stone ileus (Intermittent obstruction).

In late case, patient may present by complications, e.g. shock.

Signs

A- General:

- Dehydration:
 - ⇒ Dry tongue, tachycardia, oliguria and hypotension may be present.

B- Local:

➤ Inspection:

1. Distension of the abdomen:
 - High small IO → may be absent.
 - Low IO → mainly central (**Step-Ladder pattern**).
 - Colonic obstruction → mainly in the flanks.
2. Visible peristalsis: may be observed (absent in paralytic ileus).
3. evidences of the cause:
 - Hernial orifices:
 - Commonest cause of obstruction is external strangulated hernia (40%) ✓.
 - Scars of previous operations:
 - Obstruction by bands & adhesions (25% of cases).



Distended Abdomen

➤ Palpation:

Mass

(Empty Rt. iliac fossa)

- Sausage shaped mass with sign de Dance → intussusception.
- Volvulus mass.
- Might be palpated as in malignancy.

➤ Percussion:

- Tympanitic abdomen.

➤ Auscultation: for intestinal sounds:

- Frequent, loud in simple obstruction (Mad abdomen).
- In delayed presentation the sounds may be reduced indicating onset of secondary ileus.
- Silent in paralytic ileus, except for tinkling sound.

- DRE (normally empty):
 - Red current jelly stool or head of intussusception.
 - Carcinoma of the rectum
 - Impacted feces in the rectum (elderly bed-ridden pt).

When to suspect strangulation?

➤ Symptoms:

- Pain is not relieved by NG suction (as it is caused early by hyperperistalsis and ischemia, so decompression does not relieve ischemic pain)

➤ Signs:

- General: Toxic patient, fever or shocked according to the length of the segment.
- Local: Rigidity, marked tenderness and rebound tenderness.

➤ Investigations:

- CBC: leucocytosis.



This loop of intestine was strangulated within a hernia.

Investigations

I. For diagnosis:

▪ Plain X-Ray of the abdomen:

- Erect: multiple fluid levels. ➔ High SIO ➔ minimal fluid levels.
➔ Low SIO ➔ multiple central fluid levels.
- Supine:
 - Jejunal loop ➔ mucosal folds (Valvulae conniventes) crossing from one side of the lumen to the other & regular interval.
 - Ileal loop ➔ featureless with no mucosal pattern.
 - Colon ➔ haustrations do not reach the other side of the lumen.

▪ U/S: may show:

- a. Distended bowel loops.
- b. A mass of intussusception can also be demonstrated.

▪ Barium enema or gastrografin enema

- in colonic IO, may show:
 - a. Claw sign: in intussusception.

▪ Double enema test:

- a. 1st enema: to wash out the distal segment.
- b. 2nd enema (after ½ hour): if obstruction ➔ it will come out clear (no stool)



Multiple Fluid Levels

2. **For complications:** *to assess the general condition*

- **CBC:** hemoconcentration and leucocytosis.
- **KFTs:** Pre-renal failure.
- **Serum electrolytes.:**

Treatment

Urgent relief of obstruction after good preoperative preparation ✓

I- **Correction of general condition:**

1. **IV line:**

- Replacement of lost fluid:
 1. Fluids and electrolytes.
 2. Blood and plasma in case of shock.
- Antibiotics if there is suspicion of strangulation.

2. **NG suction to:**

- Decompress the bowel, to:
 - Relieve the pain.
 - Relieve the pressure on the intestinal wall → ↑ blood flow and preserves intestinal viability.
 - ↓ incidence of postoperative paralytic ileus.
- Reduces the risk of inhalation during induction of anesthesia.

3. **Catheter :**

- Urine output.(at least 0.5ml/Kg/hour) .

4. **Monitoring of fluid status by:**

- Pulse
- Blood pressure
- Tissue turgor

II- **Conservative treatment:**

- **Indications** in early non-complicated cases of:
 1. Adhesive intestinal obstruction, may be relieved by IV drip and NG suction.
 2. Ileocecal intussusception may be relieved by hydrostatic effect of barium enema.
 3. Sigmoid volvulus may be reduced by rectal tube.
 4. Fecal impaction → treated by enema.
- **Contraindications:** in late, complicated or doubtful cases

III- **Operation is done to relieve the obstruction:**

- **Anesthesia:** General.
- **Incision:** Usually midline, long exploratory except in strangulated hernia directly over it.
- **Exploration & determination of the level of obstruction:**

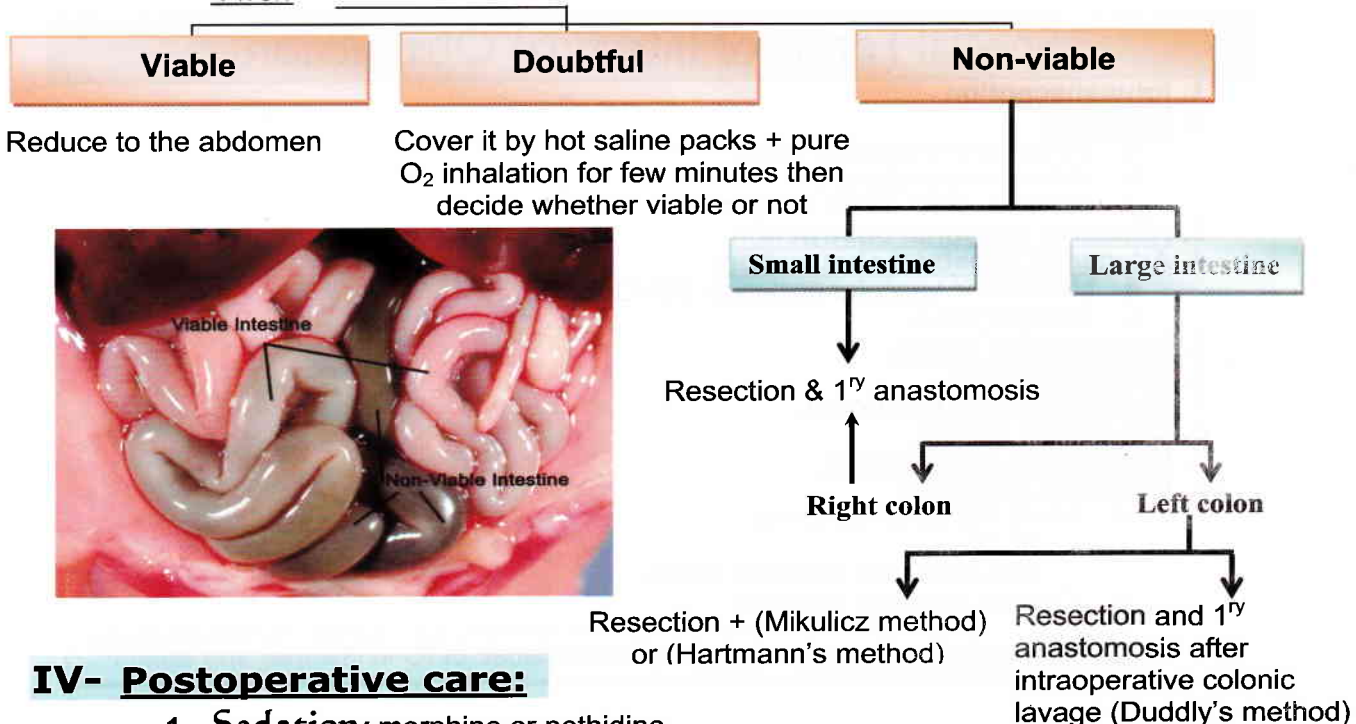
Palpate the caecum

 - If **collapsed** → small bowel obstruction → follow the collapsed ileum till you reach the site of obstruction.

- If **distended** → the obstruction in the large bowel or rectum.
- Caecum is diagnosed intraoperatively by:
 - Appendices epiploicae.
 - Tenia coli.
 - Blind pouch.
 - Bluish coloration.
- Relieve the obstruction by treatment of the cause:
 - e.g. reduction and repair of hernia or untwisting of the volvulus or division of adhesion bands
- Assessment of bowel viability:

		Viable	Not viable
Musculature	Consistency	Firm	Floppy
	By pricking	Contracts	No response
Vasculature	Color	Pink	Brown to black
	Mesenteric arteries	Pulsating	Non-pulsating
	If injured	Bleeds	No bleeding
Peritoneal covering	Luster	Present	Absent

- Then



IV- Postoperative care:

1. Sedation: morphine or pethidine.
2. NPO.
3. IV fluids:
 - 3 liters (0.5 liter saline & 2.5 liters glucose 5%) → daily requirements.
 - Additional amount of Ringer's solution equals to the volume of gastric suction.

3 Questions must be asked if IO is suspected

1- Is it obstruction?

→ Intestinal obstruction is diagnosed by colicky pain, distention, vomiting and absolute constipation

2- What is the pathological type of the obstruction (simple or strangulated)?

3- What is the level of the obstruction?

	High S.I. obstruction	Low S.I. obstruction	Colonic obstruction
Vomiting	Early & repeated → early dehydration	Delayed for 12 hours	Absent or delayed
Distention	Absent or little	Central	Marked especially in the flanks
Constipation	Absolute, may be delayed until passage of distal stools		Early

Special Forms of Intestinal Obstruction

1. Intussusception
2. Volvulus:
 - a. Volvulus of the pelvic colon.
 - b. Volvulus neonatorum.
 - c. Volvulus of the small intestine.
3. Adhesive intestinal obstruction
4. Adynamic intestinal obstruction:
 - a. Mesenteric vascular occlusion (MVO)
 - b. Paralytic ileus.
5. Strangulated hernia.
6. Meconium ileus.
7. Meconium plug syndrome.
8. Congenital atresia.
9. Necrotizing enterocolitis.
10. Intestinal ischemia:
 - a. Acute intestinal ischemia:
 - MVO
 - Non-occlusive ischemic colitis.
 - b. Chronic intestinal ischemia.

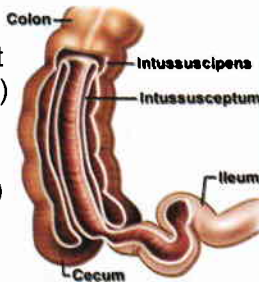
- Strangulated hernia is the commonest cause of IO in children and adults[✓].

Intussusception

1. Primary Intussusception

Definition

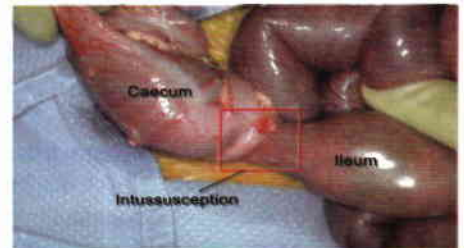
- Invagination of an intestinal segment (intussusceptum) into the lumen of an adjacent one (intussusciens) or telescoping, due to unknown etiology.



- **Etiology:** idiopathic (adenovirus??)
- Mostly ileocecal
- **C/P:** child with colicky abdominal pain + red currant jelly stool
- **important signs:**
 - 1- Sausage shaped mass
 - 2- Sign de dance
 - 3-DRE: blood and mucous
- **Invest.:** Ba enema → claw sign, U/S → target sign
- **TTT:** pre-op. preparation, conservative by hydrostatic barium reduction if failed surgery

Pathology

- An intussusception is composed of:
 - 1- Entering or inner layer. } intussusceptum
 - 2- Returning layer
 - 3- Outer sheath } intussusciens
- The blood supply of the inner layer is liable to be impaired at the neck of intussusception.
- **Ileocecal:** (75% → commonest)
 - The terminal ileum invaginates into the colon with the ileocecal valve forming the apex of the intussusception.



Etiology

Infantile intussusception (idiopathic)

- Possible cause is adenovirus → swelling of Peyer's patches (maximum aggregation at terminal ileum) which protrudes into the lumen → acts as a FB → forced distally along the gut.
- **Evidence:** frequent occurrence of intussusception at age of weaning, summer (due to increased incidence of gastroenteritis).

Clinical Picture of Infantile Ileocecal Intussusception

Any infant having colicky abdominal pain with passage of blood-stained mucus per rectum should be suspected of having intussusception until proved otherwise

Type of Patient

- Usually a healthy male between 3 – 12 months old (male: female= 2:1).
- Common at the age of weaning or following recent gastroenteritis (caused by adenovirus or rotavirus).



Ileo-cecal intussusception

Symptoms

➤ Pain:

A. In attacks:

- Characterized by:
 - Crying, screaming & drawing the legs up.
 - Pallor & lethargy.
- Between attacks: well-being child seeking for feeding.

B. If constant → suspect strangulation.

➤ Vomiting:

- Projectile vomiting following attacks of colic in 85% of patients.

➤ Absolute constipation:

- The child passes blood & mucus per rectum which is called (**Red Currant Jelly stools**).
- The mother may deny absolute constipation as she is confused by the passage of blood and mucous per anus.



Signs

➤ General:

- Dehydration: sunken eyes, depressed fontanelles & inelastic skin.

➤ Local:

▪ Inspection:

1. **Visible peristalsis** may be observed.
2. **Distension of the abdomen** (usually absent in early cases).

▪ Palpation:

1. **Sausage-shaped mass** may be felt → concave towards umbilicus. It hardens during the attack & softens in between attacks.
2. **Signe de Dance** → right iliac fossa feels empty.

▪ Percussion: Tympanitic abdomen.

▪ Auscultation: mad abdomen.

▪ DRE:

1. The finger is stained with mucous & blood.
2. Apex of the intussusception may be felt.
3. Very rarely, intussusception may protrude through the anus.

If complicated by peritonitis:

Rigidity, guarding, tenderness & Rebound tenderness

Complications and Cause of Death

- Shock & toxemia from gangrenous segment (1ry intussusception is prone to early gangrene than 2ry intussusception).

Differential Diagnosis

1. Acute enterocolitis:

- Faecal matter is present together with the blood & mucus.

2. Rectal Prolapse:

- A finger can be introduced into the rectum along the intussusception, but in prolapse the finger can not be introduced.

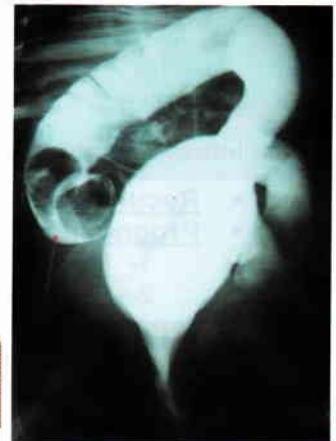
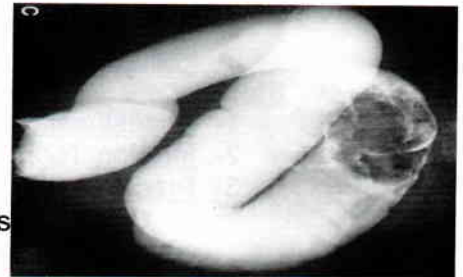
3. Henoch's purpura:

- Purpuric rash is present; it may be associated with intussusception which will need operation.

Investigations

1. For diagnosis:

- **Plain X-Ray:**
 - Erect → multiple fluid levels
 - Supine → detects the distended loop:
 - Jejunal loop → mucosal folds crossing from one side to the other
 - Ileal loop → featureless
- **U/S with saline** → Target sign
- **Barium enema (Diagnostic & Therapeutic):**
 - It shows the "Claw sign" .
 - Cylindrical filling defect



2. For complications:

(to assess the general condition of the patient)

- **CBC:** anemia
- **Serum electrolytes**
- **KFTs**

Urgent operation after preparation

Treatment

Preoperative Preparation

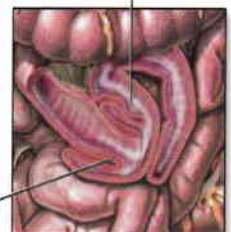
- IV fluids and antibiotics.
- NG suction.
- Catheterization.
- Monitoring.

Hydrostatic Reduction

- **Indications:** only early non complicated case using barium enema.
- **Contraindications to hydrostatic reduction:**
 - 1- Late cases (> 48 hours).
 - 2- Presence of abdominal distension or rigidity; i.e. complicated.
 - 3- Doubtful diagnosis → exploration should be done.
- **Method:** using barium enema.
Barium is running through the rectum under pressure (not more than 120 cm H₂O).
- **Success rate:** 50%.
- **Success of reduction is confirmed by:** Filling of the terminal ileum, caecum & appendix (no claw sign).



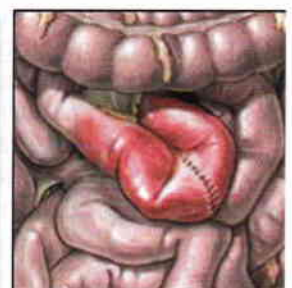
Barium traveling in affected intestine



Barium enema
Intussusception

Before

After



Surgical Reduction

- **Indications:**
 - 1- Late complicated cases from the start.
 - 2- Failure of hydrostatic reduction.
- **Steps:**
 - 1- Anaesthesia: General.
 - 2- Incision: Right paramedian.
 - 3- Procedure:
 - **Squeeze method:**
 - The mass is reduced by squeezing the apex gradually proximally (never to pull on the ileum).
 - The most difficult part to be reduced is the last part.



If the intussusceptum is irreducible or gangrenous, resection & end to end anastomosis

- **Recurrence:** avoid by stitching the terminal ileum to the caecum.
- **Prognosis:**
 - 1- Mortality: Is high in gangrenous lesions.
 - 2- Recurrence: 2% in primary intussusception.

2. Secondary Intussusception

Types

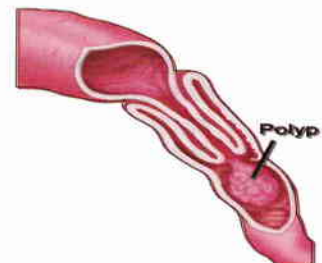
- 1- **Ileocecal:** (75% → *commonest*)
 - The terminal ileum invaginates into the colon with the ileocecal valve forming the apex of the intussusception.
- 2- **Ileoileal:**
 - A loop of ileum invaginates itself into an adjacent ileal loop.
- 3- **Ileocolic:**
 - An ileal loop invaginates into an ileal loop, and then passes to the colon through the ileocecal valve → more liable for strangulation.
- 4- **Colocolic:** A loop of colon invaginates into an adjacent colonic segment.
- 5- **Retrograde:** e.g. jejunogastric intussusception after partial gastrectomy.
- 6- **Multiple.**



Adenomatous polyp causing Intussusception in Large intestine

Etiology

- Occurs mainly in adults
- There is an evident cause as:
 1. Polyp.
 2. Submucous lipoma.
 3. Proliferative carcinoma.
 4. Inverted Meckel's diverticulum.
 5. Submucous hematoma in Henoch Schonlein purpura.



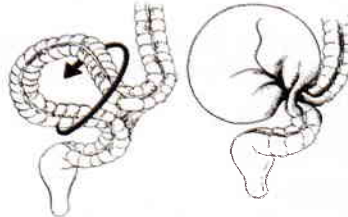
3. Volvulus

Definition

- Twisting of a loop of the bowel around an axis of its own mesentery

Sites

1. Pelvic colon.
2. Small intestine.
3. Stomach.
4. Caecum.



Pathology

- Volvulus produces a combination of obstruction of the closed loop type and occlusion of the main vessel at the base of the involved mesentery.
- In a closed loop, the pressure rises rapidly → gangrene & perforation.

A. Volvulus of the Pelvic Colon

Predisposing Factors

- Sigmoid volvulus is common in **elderly chronically constipated males** due to:
 - a. Long sigmoid colon.
 - b. Narrow base of sigmoid mesocolon.
 - c. Heavily loaded sigmoid as a result of chronic constipation.
 - d. Adhesions at the apex of the sigmoid colon facilitate its twisting.

- Common in old ♂ with chronic constipation
- C/P:** Symptoms and Signs of IO but vomiting delayed 1-2 days, tense balloon of volvulus can be felt
- Invest.:** a. OMEGA sign in X-ray.
b. Ace spade deformity in barium enema
- TTT:** pre-op. preparation, conservative by rectal tube & if failed surgical correction

Pathology

- The loop may rotate $\frac{1}{2}$ turn up to $1\frac{1}{2}$ turn which leads to:
 - Occlusion of veins → congestion.
 - Occlusion of the arteries → gangrene & perforation.

Clinical Picture

Type of patient

- Elderly constipated male with repeated episodes of abdominal pain

Symptoms

1. **Pain:** sudden severe colicky pain.
2. **Marked distension:** in the flanks from Lt. side towards the umbilicus.
3. **Constipation:** absolute but bleeding per rectum may be present.
4. **Vomiting:** delayed for 1-2 days.

Signs

1. **Inspection:**
 - Distension → early in the Lt side for 2-3 hrs → central
 - Visible peristalsis.
2. **Palpation:**
 - Tense balloon of the volvulus** can be felt → going towards umbilicus.
3. **Percussion:** Tympanitic abdomen.
4. **Auscultation:** mad abdomen.
5. **DRE:** empty rectum.

Neglected cases show evidence of peritonitis:
Rigidity, guarding, tenderness & Rebound tenderness

Complications and Causes of Death

- Shock & toxemia from gangrenous segment

DD

- **Intestinal obstruction in an old, chronically constipated male:**
 - 1- Cancer colon (progressive constipation)
 - 2- Volvulus of the pelvic colon (non-progressive)
 - 3- Diverticulitis

Investigations

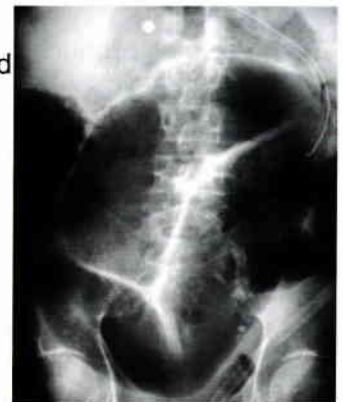
1. For diagnosis

- **Plain X-Ray of the abdomen:**
 - **Omega sign (Ω)** $\square$: There will be a very big loop distended with gas & occupying nearly the whole abdomen.
- **Barium enema:**
 - **Ace of Spade deformity ($\spadesuit$)** $\square$: (distended rectum) with arrest of barium at rectosigmoid junction.



2. For complications:

- **CBC:** anemia.
- **KFTs:** pre-renal failure.
- **Serum electrolytes.**



Treatment

1. Preoperative preparation "resuscitation"

- NG suction "Ryle".
- IV line
- Catheter.
- Monitoring.

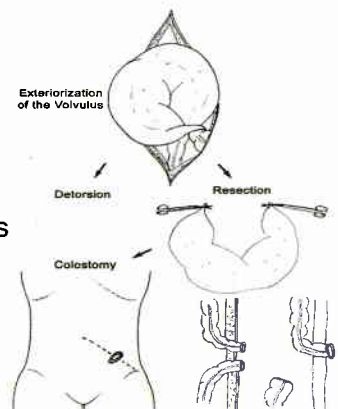
2. Conservative Treatment

- **Indications:**
 - Early non-complicated cases (i.e. no evidence of gangrene).
- **Method:**
 - A **rectal tube** $\square$ is passed through a sigmoidoscope to untwist the sigmoid loop.
 - If successful: **the tube is left in place** and the patient is prepared for later elective resection of the long sigmoid to prevent recurrence.
- **Success is confirmed by:** gush of gases & fluid stools.



3. Surgery

- **Indications:**
 - Failure of the conservative treatment.
 - Late complicated cases.
- **Method:**
 - 1- If the colon is viable and short $\rightarrow$ Fix to the posterior abdominal wall (colopexy)
 - 2- If the colon is viable & long $\rightarrow$ Mikulicz or Hartmann's method.
 - 3- If the colon is gangrenous $\rightarrow$ Mikulicz or Hartmann's method.

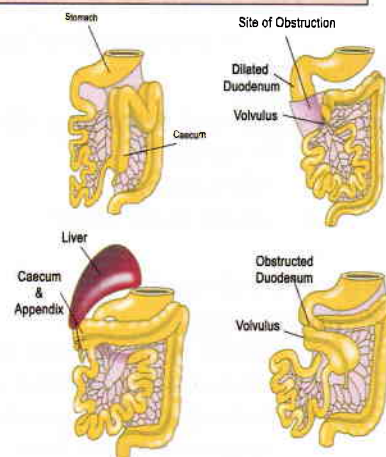


4. Postoperative care

- NPO.
- Analgesics.
- Sedatives.
- IV fluids.

B. Volvulus Neonatorum

- The condition is caused by incomplete rotation of the midgut that should occur during intra-uterine life.
- The baby is born with a free midgut having a narrow-based mesentery, and is, therefore, liable to twist around its mesenteric axis. The result is strangulation obstruction of the whole midgut.
- In addition, there is usually a fibrous band that extends from below the liver to the left to the caecum which lies in the epigastrium. This can obstruct the duodenum.
- Urgent surgery is needed to untwist the gut, to divide the band and to broaden the base of its mesentery by further displacement of the caecum to the left.



4. Adhesive Intestinal Obstruction

- Intraperitoneal adhesions constitute one of the **commonest** (50%) causes of small intestinal obstruction.

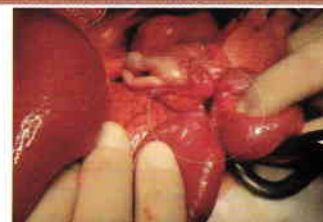
Pathology

- Adhesions may be solitary or multiple.
- They bind one intestinal loop to another.
- Obstruction results from:
 - Kinking
 - Direct obstruction of the small intestinal loop.
- Strangulation may result from:
 - Compression of the loops' blood supply
 - A band may induce localized ischemic necrosis by direct pressure on the intestine at the level of block.
- Adhesions have a tendency for recurrence producing recurrent intestinal obstruction.

- ▶ It is the commonest cause of IO in adult
- ▶ **Etiology:** postoperative, post-inflamm., trauma
- ▶ **C/P:** IO + cause e.g. scar or H/O of operation
- ▶ **TTT:** Mainly conservative, if failed or strangulation surgical TTT is required

Causes of Adhesions

- Post-operative (most common) especially appendicitis & gynaecological operations
- Post-inflammatory: e.g. septic or TB peritonitis.
- Foreign material - particularly talc, silk thread, barium sulphate and asbestos.
- Trauma.
- Vascular occlusion.



Clinical Picture and Investigations

*The same as in other causes of acute intestinal obstruction.
+ presence of scar or history of previous operations*

Treatment

- The treatment of adhesive obstruction is mainly conservative :

1. IV fluids.
2. NG suction.
3. Close observation.

- **Surgical treatment:**

➤ indications:

- There is suspected strangulation (tenderness, rebound tenderness and rigidity).
- Failure of conservative treatment e.g. no response more than 72 hours:

➤ Steps:

- 1) Resuscitation & Monitoring.
- 2) Exploration & division of adhesions (cut only the causative adhesion).
- 3) Cover any serosal tear.
- 4) Deal with the intestine (If gangrenous).

➤ Postoperative care:

- Sedation, NPO, IV fluids

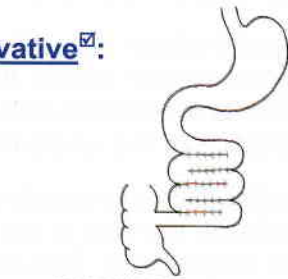


Figure 66.12 Noble's plication.

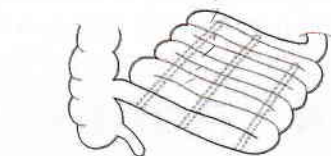


Figure 66.13 The Charles-Phillips procedure.

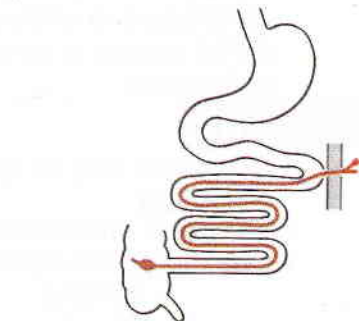


Figure 66.14 A Baker's tube inserted via a Witzel jejunostomy may also be used.

5. Adynamic Intestinal Obstruction

A. Mesenteric Vascular Occlusion

Definition

- It is occlusion of the superior (or rarely the inferior) mesenteric vessels or one of their branches.

- ▶ **Etiology:** arterial or venous occlusion
- ▶ **C/P:** young adult (AF) or elderly (atherosclerosis)
IO + Strangulation, other system affection
If non-occlusive: ↑ frequency of pain, ↓ BP
- ▶ **Invest.:** mesenteric angiography, duplex, X-ray
+ cause, comp., D.D.
- ▶ **TTT:** pre-op. preparation + urgent laparotomy then deal acc. to the state of the intestine {Healthy or Gangrenous} + TTT of the cause

Pathology

- **Arterial obstruction** → ischemia of the mucosa → ulceration and sloughing in 3 hours → intestinal bacteria cross the mucosal barrier → peritonitis, after 6 hours gangrene occurs (bacterial translocation).

- **Venous obstruction** (on top of portal hypertension or collagen disease) → gangrene is more delayed than arterial obstruction.
- **Reperfusion damage** → the return of blood flow (from gangrenous ischemic segment) containing bacterial toxins & oxygen free radicals from this segment → to systemic circulation

▪ Whether the occlusion is arterial or venous, the infarction is always a red hemorrhagic infarction.

- **Non-occlusive ischemia** : inadequate perfusion due to hypotension, spasm or intestinal distension in patients with underlying atherosclerosis.



Causes

1. **Arterial embolism**☐: (source: MS + AF, MI + Mural thrombus).
2. **Arterial thrombosis**: due to atherosclerosis (with history of intestinal angina), more insidious onset than embolism.
3. **Venous thrombosis**: due to portal hypertension, hypercoagulable state, intraperitoneal sepsis and contraceptive pills.

Clinical Picture

Type of Patient

- It is common in the elderly due to thrombosis (on top of atherosclerosis).
- It may occur in young patients with source of embolism e.g. AF.

Symptoms

Manifestations of (acute IO + strangulation) ☐

1. Pain: severe (acute abdomen) (out of proportion to physical signs and not relieved by NG suction).
2. Bleeding per rectum when mucosal infarction has occurred.
3. Vomiting and diarrhea may be present.
4. In late cases patient may present with shock and toxemia.
5. Non-occlusive ischemia should be suspected in increased frequency and abdominal pain following prolonged hypotension period among critically ill patients in ICU with low blood flow state.

Signs

If neglected

▪ General:

-Shock and toxemia

▪ Local

-Guarding, tenderness and rebound tenderness.(peritoneal irritation; with full thickness infarction)

DD

1. Acute pancreatitis: serum amylase ↑ in 5% of pts with MVO (but not reaching levels as high as those of acute pancreatitis).
2. Intestinal strangulation.

Complications & Causes of Death

If long segment → hypovolemic shock

If short segment → toxemia

Investigations

There is no specific test; the only key for diagnosis is **to bear the condition in mind** especially if the history is suggestive e.g. young age with AF or old age with atherosclerosis.

1. For diagnosis:

- **Mesenteric angiography or duplex scan:**
 - The value is controversial as it may delay surgical interference.
- **Plain X-Ray:**
 - Multiple fluid levels.
 - Late cases: intestinal necrosis (intraluminal & intramural gas).
- **CT Scan.**



CT scan of MVO

2. For complications: to assess the general condition of the patient

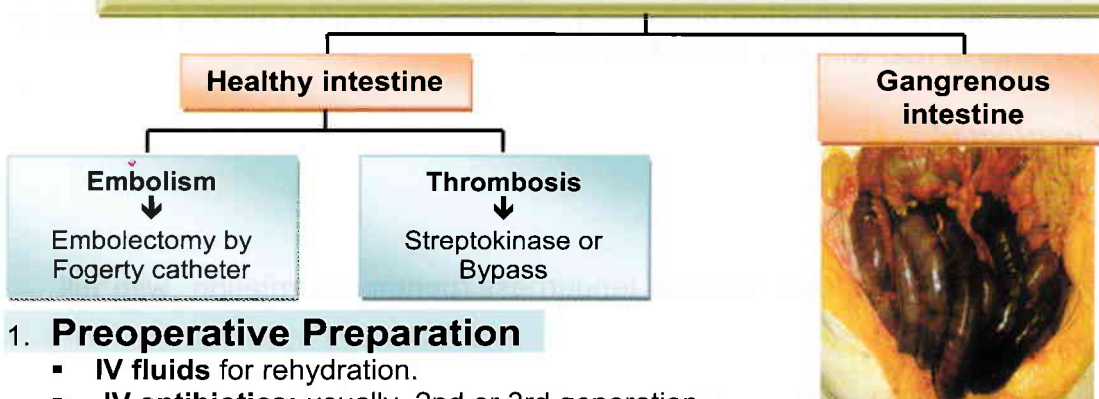
- **CBC:** ↑ TLC may be used to monitor progress of the disease.
- **KFTs:** pre-renal failure.
- **Increased serum amylase**
- **Serum phosphate level:** may be elevated but not specific for bowel ischemia.
- **Metabolic acidosis:** late and suggests bowel necrosis.

3. For the cause e.g. ECG or echo, US (routine in cases of acute abdomen)

4. To exclude pancreatitis e.g serum amylase

Treatment

Resuscitation & Urgent laparotomy to explore the patient



1. Preoperative Preparation

- **IV fluids** for rehydration.
- **IV antibiotics:** usually 2nd or 3rd generation cephalosporins together with metronidazole.
- **Correction of metabolic acidosis.**
- **Heparin:** continuous infusion for thrombo-embolic disease to prevent clot extension and DIC. it is interrupted during surgery.

Gangrenous Intestine

2. Urgent Operation

I. Gangrenous intestine:

- Resect the gangrenous part.
- Primary anastomosis is better avoided in case of:
 - 1- Peritonitis with friable tissue.
 - 2- Doubtful viability of the remaining intestine.
 - 3- Patient with bad general condition.
- For patient who received 1st anastomosis, a 2nd look operation is recommended after 24 hr.

2. Viable intestine:

- Mesenteric arterial occlusion: Embolectomy, thromboendarterectomy or bypass graft then post-operative anticoagulants for 3 months.
- Mesenteric venous thrombosis: Diagnosed at operation by edema of mesentery and extrusion of clots from mesenteric veins when they are cut.
- **Treated by:**
 - Resect gangrenous intestine, give postoperative heparin and continue oral anticoagulants for at least 3 months.

3. Postoperative care

- The patient is given postoperative heparin and discharged under oral anticoagulants for at least 3 months to prevent recurrence.
- Sedation, NPOI, IV fluids

4. Treatment of the Cause

- e.g. Treatment of AF, atherosclerosis.

Resection of > 70% of the intestine → short bowel syndrome.

Prognosis

Mortality rate of:

- Mesenteric venous occlusion: 30%
- Mesenteric arterial occlusion: 45%
- Mesenteric thrombosis > mesenteric embolism

➤ Acute intestinal ischemia, may be 2 types:

1. Acute MVO.
2. Non-occlusive intestinal ischemia (discussed later).

B. Paralytic Ileus

Definition

- It is failure of neuromuscular mechanism leading to failure of peristaltic waves of the intestine (functional I.O.) with patent lumen.

Etiology

- Reflex inhibition of intestinal motility** → After
 - Major abdominal operation. (**most common**)
 - Labor.
 - Retroperitoneal haematoma.
 - Fracture spine.

→ **This will result in reflex sympathetic stimulation.**
- Toxic inhibition** → Due to peritonitis, typhoid.
- Metabolic abnormalities** → due to hypokalemia, uremia and DKA.
- Drugs** → overdose of Anticholinergic or TCA.

- Paralysis of SI = paralytic ileus.
- Paralysis of stomach = acute gastric dilatation.
- Paralysis of colon = colonic pseudo-obstruction in old diabetic pt (**Ogilvie**).

- **Etiology:** reflex e.g. 3rd postoperative, toxic, metabolic, drugs
- **C/P:** IO but no abdominal pain & vomiting is effortless
Scar, no peristalsis, pseudo-shifting dullness
- **Invest.:** X-ray, for the cause
- **TTT:** **a- prophylactic:** avoid the cause
b- active TTT → TTT of the cause, R&M

Pathology

- The most affected part is ileum, so it is so named.
- Failure of transmission of peristalsis.
- Accumulation of gas & fluid inside intestine → distension.
- This distension causes more paralysis & so on.

Clinical Picture

3rd day Postoperative

Symptoms

- Distension:**
- Constipation:**
 - Absolute (except if flatus forced passively by hand per abdomen).
- Vomiting:**
 - Repeated effortless (or may be manifested by ↑ gastric aspiration if a Ryle's tube was inserted).
- Pain:** No abdominal pain except at site of abdominal incision (only sense of fullness and discomfort never colicky).

Signs

- **General:** shock + C/P of the cause, e.g. uremia, hypokalemia.
- **Local:**

- Inspection:**
 - Distension.
 - No visible peristalsis.
 - Scar of previous operation.

2. Percussion:

- Tympanitic abdomen.
- Pseudo-shifting dullness.

3. Auscultation: ▢

- Dead silent abdomen.
- High pitched tinkling abdominal sound may be heard.
(due to the passage of fluid from one distended loop to the next).

If symptoms persist beyond the 3rd day postoperative → suspect other causes e.g. adhesive intestinal obstruction or peritonitis.

Investigations

1. Radiological:

- Plain X-ray: it shows multiple fluid levels.

2. Laboratory: to assess general condition of the patient and to detect the cause

- CBC
- KFTs
- Serum electrolytes (K⁺ level, FBS).



Postoperative adynamic ileus

DD

Other causes of I.O.

Treatment

Prophylaxis (avoid the predisposing factors)

1. Preoperative → correction of the electrolyte imbalance.
2. Intraoperative → gentle manipulations.
3. Postoperative → NPO till intestinal peristalsis is regained (some surgeons prefer early fluid intake "enhanced recovery program")

Active Treatment

A- Treatment of the cause

e.g. Drainage of peritonitis, TTT of internal hemorrhage, hypokalemia.
Parasympathomimetics (prostigmine) in resistant cases (controversial)

B- Resuscitation and monitoring: (vital data, signs of recovery)

1. Ryle's tube → GIT suction to relieve the distension.
2. IV line:
 - Restoration of fluid & electrolyte balance.
 - Antibiotics.
3. Sedation → Pethidine 100 mg IM.
4. Observation for the signs of recovery, which are:
 - Wind pain.
 - Passage of flatus.
 - Distension decreases.
 - Aspiration decreases.

Post-operative care

1. Sedation
2. NPO
3. IV fluids

➡ If postoperative ileus is prolonged, you should think of :

- 1- Peritonitis from leaking intestinal anastomosis.
- 2- Mechanical obstruction from early fibrinous adhesions.

DD of Early post operative small bowel obstruction

■ **Etiology:**

1. Paralytic ileus
2. Post operative adhesions,
3. Internal hernia,
4. Peritoneal defect,
5. Intra abdominal abscess

→ Strangulation of the bowel in post operative obstruction is uncommon.

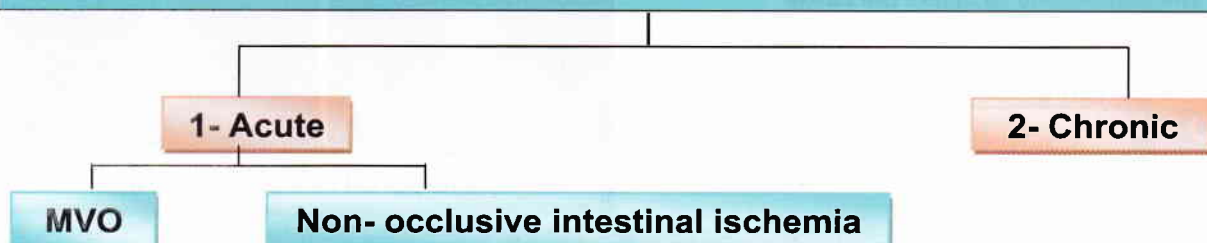
■ **Investigation:**

- X-ray → presence of gases throughout small and large intestine suggests paralytic ileus
 → Diluted Ba or gastrografen follow through → where the diagnosis is uncertain.

■ **Treatment :**

- 1- Most cases settle with non operative management
- 2- Exploratory operation if: complete obstruction ,intra abdominal sepsis or prolonged obstruction

6. Intestinal Ischemia



1. Acute Intestinal Ischemia

Mesenteric Vascular Occlusion (MVO)

See before

Non-occlusive Ischemic Colitis

Cause

- Sudden ↓ of cardiac output due to any cause as arrhythmia → splanchnic vasoconstriction
 → ischemia without obstruction although blood vessels may be atherosclerosed.
 e.g. shock and vasoconstrictor drugs.

Clinical Picture

- Patients 50 – 70 years old with sudden drop of pulse due to any cause → acute abdomen + bleeding per rectum.

Investigations

▪ **Plain X-ray:**

- Thumb prints ☑ (edematous mucosa surrounded by gas).

Treatment

▪ **Conservative**

- By IV fluids, Antibiotics, analgesics.

▪ **Surgery:**

- If gangrenous or stricture.



2. Chronic Intestinal Ischemia

Causes

1. Usually atherosclerosis
2. Rarely compression of celiac axis by median arcuate ligament of diaphragm.

Clinical Picture

- 1- **Post-cibal angina** (starts 15 – 30 minutes after meal for 1 hour).
- 2- Patient afraid to eat (loss of weight).
- 3- Abdominal bruit at upper abdomen.
- 4- Other manifestations of atherosclerosis in blood vessels.

Investigations

- **Arteriography:**
 - Diagnostic.
- **Barium:**
 - Stricture.
- **Exclude other causes of chronic abdominal pain:**
 - By GIT endoscopy.

Treatment

- 1- **Atherosclerosis:** treated by endarterectomy or bypass graft.
- 2- **Compression by median arcuate ligament:** treated by cutting of the ligament.

Neonatal Intestinal Obstruction

Etiology

A. Dynamic:

- Lumen:
 1. Meconium ileus.
 2. Meconium plug syndrome.
- Wall:
 1. Congenital atresias:
 - Esophageal atresia, duodenal atresia, jejunal atresia, ileal atresia, colonic atresia.
 2. Annular pancreas.
 3. Congenital megacolon.
 4. Imperforate anus.
- Outside the wall:
 1. Volvulus neonatorum.
 2. Irreducible congenital hernia.

B. Adynamic:

- Necrotizing enterocolitis and septicemia.

- ▶ **Etiology:** *a- dynamic:* (lumen, wall, outside the wall) *b- adynamic:* N.enterocolitis, septicemia
- ▶ **C/P:** IO + C/P of the cause
- ▶ **Invest.:** X-ray + for the cause
- ▶ **TTT:** pre-op. preparation + TTT of the cause

Clinical Picture

➤ Common for all causes

Symptoms

1. Vomiting and dehydration.
2. Colicky abdominal pain.
3. Absolute constipation.

Signs

1. Abdominal distension.
2. Visible peristalsis

➤ According to the cause

1- Volvulus neonatorum: bleeding per rectum.

2- Imperforate anus.

General examination:

To exclude associated congenital anomalies (VACTERL syndrome).

Examination of the perineum:

- Look for the presence of anus, its size and site
- Look for the presence of an anal dimple
- If there is an impulse on crying at the site of the anus, it denotes a low anomaly.
- In ectopic anus, there is a subcutaneous fistulous track full of meconium.

3- Hirschsprung's disease:

Symptoms:

- Delayed passage of meconium.
- Defecation occurs every few days.

Signs (DRE):

- Empty rectum.
- Grips on the finger.
- Characteristic gush of stool on withdrawal of the finger.

Investigations

▪ Plain X-Ray:

- Erect: multiple fluid levels.
- Supine: shows the level of obstruction.

▪ According to the cause:

a. Hirschsprung's disease:

- Barium enema: narrow aganglionic segment with proximal colon dilatation.
- Rectal biopsy: absence of ganglion cells.

b. Imperforate anus:

- Invertogram: the distance between coin & distal gas shadow is measured:
 - If < 1 cm → low anomaly
 - If > 1 cm → high anomaly
- IVP: to exclude associated urinary tract anomalies.



Double bubble sign
(duodenal atresia)

Treatment

Preoperative preparation

- IV fluids & electrolytes.
- NG suction.
- Catheter: to monitor urine output.

Operation

- **Time**: 10 months, 10 Kg & Hb 10 gm/dL
- **Procedure**: according to the cause:
 - Hirschsprung's disease:
 - Excision of aganglionic segment with anastomosis of normal colon with lower $\frac{1}{2}$ of the anal canal.
 - Imperforate anus:
 - 1. High anomalies:
 - ⇒ Treated with staged surgery:
 - First stage: Temporary colostomy
 - Second stage: Anorectal pull-through (abdominal approach)
 - Third stage: closure of colostomy.
 - 2. Low anomalies:
 - a- Covered anus:
 - Excision of skin bar.
 - b- Membranous anus:
 - Cruciate incision of the membrane with trimming of edge.
 - c- Ectopic anus:
 - Cut-back operation.
 - d- Anal stenosis:
 - Dilatation.

Section C

The Large Intestine

Colon & Rectum



Principles of Colon Surgery

- Colonic anastomosis is more liable to disruption, leakage and peritonitis more than small intestine due to:
 1. The high bacterial count of both aerobic and anaerobic organisms.
 2. Constant gaseous distension.
 3. Incomplete serous coat.
 4. The terminal arteries poorly connect with each other.

Preoperative Preparation for Elective Surgery

1- Improving nutritional status.

2- Bowel preparation:

- The risk of anastomosis leakage and wound sepsis is reduced if the large bowel is empty at the time of resection and if the bacterial flora of the colon is reduced.

a- Mechanical preparation:

○ Standard preparation:

- Low-residue diet for 4 days before surgery.
- Enemas and laxatives for 2-3 days before surgery.

○ Rapid preparation:

- Alternative method can be performed one day before surgery.
- Whole gut irrigation using 2-4 liters / hour of a balanced crystalloid solution passed via NG tube until the patient passes clear fluid per rectum.

This method is not used in cardiac or renal patients

b- Chemical preparation:

- Intestinal antiseptics (neomycin and metronidazole) for 2 days before operation.
- Systemic parenteral antibiotics: given before surgery and are continued postoperatively for 1 day.
- It is a combination of cephalosporin or an aminoglycoside with Metronidazole.

Operative Procedure

• Resection:

- The extent of resection is governed by vascularity and by disease extent.
- In radical surgery for malignancy, it is essential to remove the draining lymphatics & LNs.

• Anastomosis:

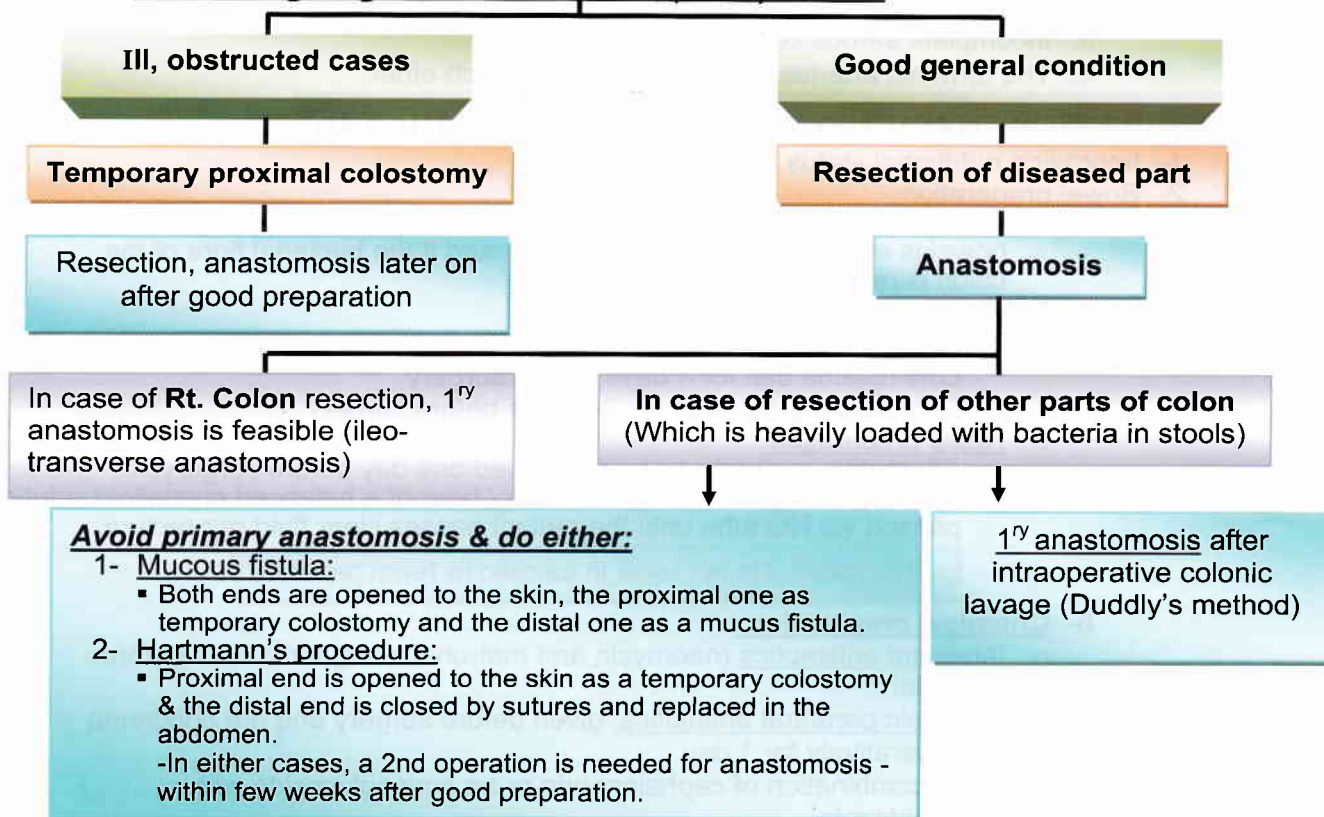
- The 2 bowel ends should be:
 - Adequately vascularized
 - Sutured without tension.
 - Water-tight & gas tight.
- An intestinal anastomosis can be done by one of two methods:
 1. Hand suturing:
 - Is commonly done in 2 layers of interrupted suture.
 - The first layer (whole through): includes the whole wall thickness.
 - The second layer (inverting Lambert): includes the serosa and muscle only.
 2. Mechanical staplers: They are faster but more costly.

Emergency Surgery

➤ Indications:

- IO, perforation, toxic dilation or massive bleeding from the colon

➤ According to general condition of the patient:

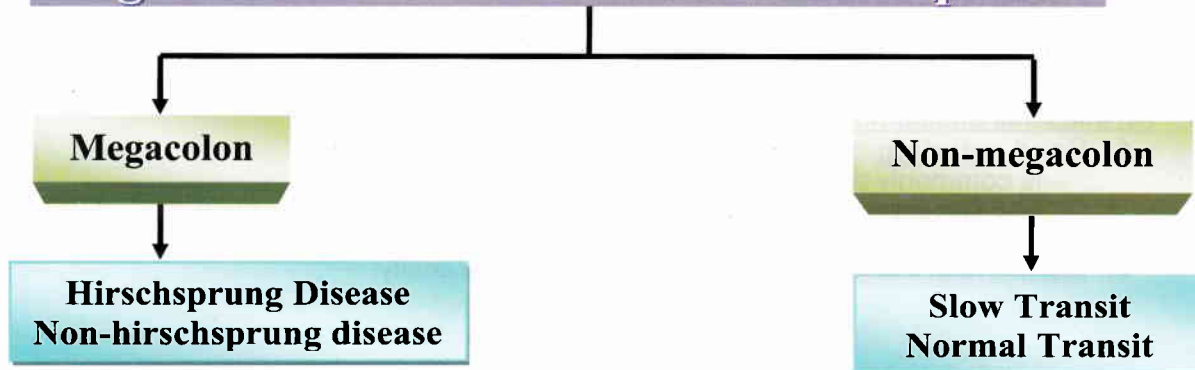


Chronic Constipation

Definition

- Bowel habits less than 1 in every 3 days (controversial)

Surgical classification of chronic constipation



Congenital Megacolon (Aganglionic Megacolon) (Hirschsprung Disease)

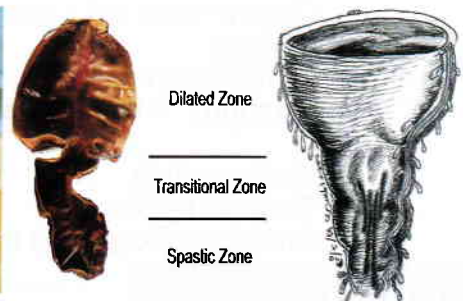
Incidence

- 1 / 5000.
- M : F = 4 : 1.

Etiology

- Unknown.
- Familial[□] in 6 % of cases.
- Associated with chromosomal disorders (e.g. Down's syndrome in 10 % of cases).
- Whatever the mechanism , it leads to failure of migration of vagal neural crest cells into developing gut leading to absence of ganglion cells

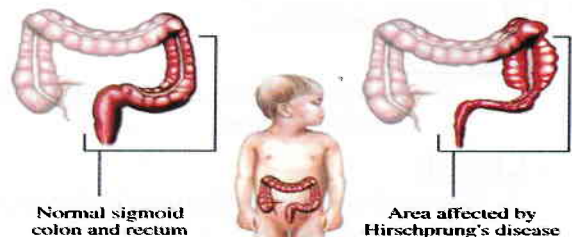
- **Etiology:** unknown, familial (6%), associated with chromosomal disorders (10%)
- **C/P:** Delayed passage of meconium > 24 hrs
Doughy mass
DRE: grips on finger, gush of foetid stool
- **Comp.:** obstructive toxic enterocolitis (cause of death), delayed growth, chest infection. Acute IO
- **Invest.:** Barium enema, rectal biopsy[□]
- **TTT:** a. Patient with obstructive toxic enterocolitis: Conservative TTT & if failed urgent colostomy
b. Patient without IO → elective Swenson operation after preparation



Pathology

- The disease starts in the upper part of the anal canal and extends proximally to the recto-sigmoid area in 80% of cases (typical cases) or it may involve variable lengths of the colon or even the whole colon.
- Severity of the case is related to absence of acetyl choline esterase rather than the length

- Macroscopically There are 3 segments:



Spastic segment	Transitional one	Proximal dilated one
Absent ganglia	Few ganglion cells	Normal ganglion cells
Collapsed	Conical	Dilated

- Microscopic:
 - Absence of ganglia[□] of Auerbach's and Meissner's plexus.
 - Hypertrophic nerves in distal large intestine.

Clinical Picture

Any newborn presenting with delayed passage of meconium for more than 24 hrs should be considered as having Hirschsprung's Disease until proved otherwise

I. The most common presentation:

Symptoms

Chronic constipation:

- Delayed passage of meconium > 24 hrs[☒].
- Defecation occurs every few days & only after insertion of a finger of the mother in the anus of the baby.

Signs

1. Inspection:

- Abdominal distension, visible peristalsis.

2. Palpation:

- Doughy mass in Lt. iliac fossa.

3. Percussion:

- Tympanitic resonance.

4. Auscultation: ↑ intestinal sounds.

5. DRE:

- Empty rectum
- Grips on the finger.
- Characteristic gush of foetid stool on withdrawal of the finger[☒].

Complications

➤ Of severe cases :

General:

1. Delayed growth and development.
2. Chest infection.

Local:

- 1- Obstructive toxic enterocolitis (fever, diarrhea and abdominal distension)

→ **Cause of death.**

- 2- Acute obstruction.

➤ Of moderate cases : failure to thrive

Investigations

I. Imaging:

▪ Plain X- ray:

- Large bowel obstruction (haustrations do not reach the other side of the lumen.
- In toxic enterocolitis perforation → air under diaphragm.

➤ The most important diagnostic tools are[☒]:

- Barium enema
- Rectal biopsy

Barium Enema:

Precautions:

- Without preparation[☑], as preparation will mask changes in colonic caliber.
- Mixed with saline (light barium) to avoid intestinal obstruction.
- No DRE before the test[☑].

Finding:

- Will show narrow aganglionic segment with marked proximal colon dilatation.
- Its accuracy is 80%.



2. Instrumental:

Rectal biopsy: (most diagnostic[☑])

- Shows absence of ganglion cells.
- Previously: full-thickness rectal biopsy
- Nowadays: small segment of mucosa and submucosa is taken by special forceps (or suction biopsy).

Anorectal manometry:

- Reveals failure of relaxation of anal sphincters in response to rectal distension.

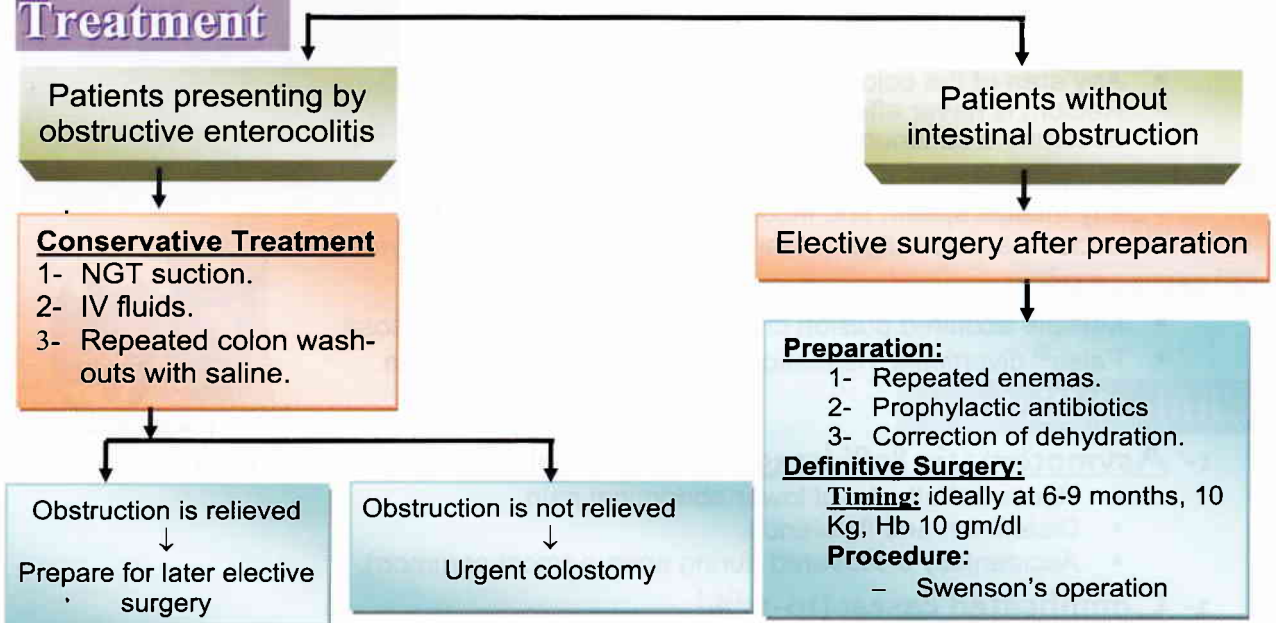
3. Laboratory:

to assess general condition.

- CBC: high TLC, hemoconcentration.

- KFT: pre-renal failure

Treatment



Operative Details

Swenson operation:

- It entails complete excision of the aganglionic segment up to 4 inches above the funnel and then anastomosis of the normal colon to the lower half of the anal canal.

Diverticular Disease of the Colon

- Diverticular diseases of the GIT include diverticular diseases of the colon & small intestine.

Incidence

- Common in western countries (50% above 65 years).
- Urban areas > rural areas.
- In Egypt it is less common and is seen more in younger age groups.

Etiology

- Chronic constipation due to low fiber diet.

Pathogenesis

- Chronic constipation → ↑ intraluminal colonic pressure → muscle spasm and incoordination.
- Later on, herniation of colonic mucosa through the circular muscle layer between the taenia coli at the points of entry of blood vessels (pulsion diverticula).

Pathology

Site

- Sigmoid colon in 90% of cases.
- Any area of the colon may be involved.
- Rectum is never affected (as taenia coli becomes a continuous layer).

Macroscopic

- Early muscle spasm and incoordination occurs. Next, the mucosa starts to bulge out through the circular muscle layer.

Microscopic

- Multiple acquired pulsion diverticula of the colonic mucosa.
- False diverticula: herniation of mucosa and submucosa.

Clinical Picture

1- Asymptomatic 80% (stage of diverticulosis) or complains of:

- Recurrent attacks of lower abdominal pain.
- Distension and flatulence.
- Accidentally discovered during enema (most common).

2- Complicated cases: (10-25%)

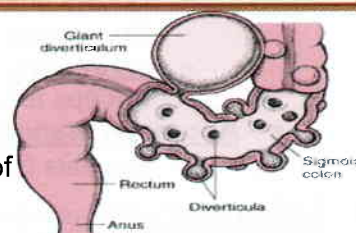
A- Acute diverticulitis:

- As acute appendicitis but on the left side (pyrexia, vomiting, generalized abdominal pain that shifts and becomes localized to the left iliac fossa, tenderness and guarding in the Lt. iliac fossa)

B- Chronic diverticulitis:

- Long history of:
 - Recurrent attacks of abdominal pain.
 - Passage of blood and mucous per rectum.

- **Etiology:** chronic constipation
- **Path:** mostly in sigmoid colon, never in rectum false & not precancerous
- **C/P:** Mostly Asymptomatic (80%), comp.
- **Comp.:** Acute & chronic diverticulitis
Massive bleeding per rectum
- **Invest.:** Ba enema (saw-tooth), sigmoidoscopy
CT in acute diverticulitis, angiography in bleeding
- **TTT:** uncomplicated cases: high fiber diet & anti-spasmodics + TTT of complications if present



Colon Diverticula

- On palpation:
 - Tender mass in the Lt. iliac fossa.

C- Bleeding per rectum:

- May be the first presentation
- Massive bright red bleeding
- Usually stops spontaneously.
- Approximately 50% of diverticular bleeding originates in the Rt. Colon despite the low incidence of diverticula in it.

Complications

1. Acute diverticulitis: (2ry to obstruction of its neck) and its complications.

▪ Perforation:

- If > 48 hrs → localized peritonitis & diverticular abscess (palpable tender mass in the left iliac fossa).
- If < 48 hrs → generalized purulent or faecal peritonitis.

▪ Fistula formation:

- Vesico-colic or vagino-colic.

2. Chronic diverticulitis: → colon stricture and adhesive intestinal obstruction.

3. Bleeding per rectum due to:

- Erosion of blood vessel at the neck of a diverticulum by inspissated faeces.
- Associated angiomatous malformation.

DD

- 1- Cancer colon.
- 2- Angiodysplasia of the colon.

It is not precancerous.

The most common causes of massive bleeding per rectum in old age are diverticular disease of the colon & angiomatous malformations. Colonic cancer is a common cause of fresh bleeding per rectum but not massive bleeding.

Investigations

The most important diagnostic tool is:

- Barium enema

I- Radiological:

▪ Barium enema:

- Early:
 - Saw-tooth appearance
- Late:
 - Well-developed diverticula can be visualized.



Barium enema and colonoscopy are contraindicated in acute diverticulitis as they can cause perforation.

▪ CT scan:

- Best investigation in acute diverticulitis.
- It reveals thickening of colonic wall, pericolic masses and abscesses
- Can show colovesical fistula with air in the urinary bladder.

▪ Angiography:

- In case of bleeding per rectum to localize the site of bleeding.



Barium Enema

2- Instrumental:

▪ Sigmoidoscopy:

- Will detect the mouths of diverticula.
- Its main value is to exclude carcinoma.
- Can detect bleeding from diverticula

Treatment

1- Treatment of uncomplicated cases:

1. High-fiber diet.
2. Anti-spasmodics for the abdominal colic.

2- Treatment of complicated cases:

A- Acute diverticulitis:

- Hospital admission.
- Bed rest, bowel rest.
- Antibiotics.
- Regular monitoring.

1. If complicated diverticular abscess:

- Incision and drainage in huge multilocular abscess.
- Pericolic abscess: percutaneous U/S guided drainage, then resection in patients who remain symptomatic or who develop recurrent abscess.

2. Generalized peritonitis:

- Resuscitation and urgent laparotomy.
- Peritoneal toilet and drainage.
- Resect the perforated colon by a Hartmann's procedure.

3. colo-vesical Fistula:

- Resection with repair of the fistula.

B- Chronic diverticulitis.

- Colectomy after proper preparation of the colon.

C- Bleeding per rectum:

- Resuscitation followed by angiography or Tc99 to localize the site of bleeding:
- If localized → regional resection
- If localization fails → total colectomy + ileorectal anastomosis.



		Diverticular Disease of the colon	Carcinoma of the pelvic colon
Symptoms	Bleeding per rectum	Profuse and periodic	Small amount and continuous
	Pain	Common	Absent in 25 % of cases
	Duration	Long	Short
Signs	Mass	Usually tender	Usually not tender
Investigations	Barium Meal	Saw-tooth appearance	Stricture, shouldering & apple core appearance
	Sigmoidoscope	Shows the mouths of diverticulum	Visualizes the mass and takes biopsy

Inflammatory Bowel Diseases

- The term “**Inflammatory Bowel Diseases**” is usually used to denote ulcerative colitis and Crohn’s disease. However, other diseases, e.g. bilharzial colitis, amebic colitis, ileocaecal TB and typhoid enteritis can cause bowel inflammation.



1- Ulcerative Colitis

Definition

- An inflammatory disease of the colon characterized by ulceration.

Incidence

- Female > Male[☐].
- In 4th and 5th decades.

Etiology

- *Unknown*
- May be autoimmune, genetic or environmental.

Pathology

Site

- Confined to the rectum (proctitis), Lt. sided colitis or whole colon and rectum (pancolitis)
- Rarely affects terminal ileum (if ileocaecal valve is incompetent → backwash ileitis[☐]).

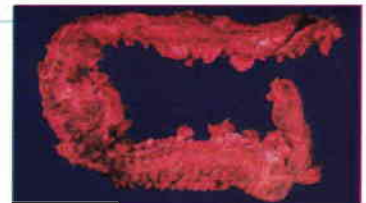
- ▶ **Etiology:** unknown, may be autoimmune
- ▶ Commonly affect the rectum and colon.
- ▶ **Path.:** confined to mucosa and submucosa with no skip lesions
- ▶ **C/P:** Watery diarrhea, Abdominal pain, wt. loss
- ▶ **Comp.:** general, local (toxic megacolon, malig.)
- ▶ **Invest.:** Ba enema, colonoscopy & biopsy[☐]
- ▶ **TTT:** Medical: rest, diet, anti-inflammatory
Surgical: panproctocolectomy + ileal reservoir



Different degrees of ulcerative colitis

Macroscopic Picture

- Varies from mucosal edema to extensive ulceration
- No skip lesions[☐] (which are a feature of Crohn’s disease)



Microscopic Picture

- The inflammation is confined to mucosa and submucosa[☐] (superficial).
- Serosa is usually normal[☐].
- Characteristic histological feature is: Crypt abscesses[☐] with surrounding inflammation and pseudopolyps[☐] (edematous mucosa surrounded by ulcers[☐] and they are highly precancerous and considered an indicator of total proctocolectomy).

Clinical Picture

- 1- Watery diarrhea + (blood, pus, mucous and tenesmus).
- 2- Abdominal pain.
- 3- Weight loss and dehydration.
- 4- May present with complications.
 - e.g. **Toxic megacolon:**
 - This is a very serious complication.
 - Severe diarrhea with passage of blood, mucous, pus with stools.
 - Patient is very toxic with severe pyrexia 39 – 39.5°C.

- Severe abdominal distension due to marked colonic atony.
- Unless treated urgently, it has a fatal prognosis.

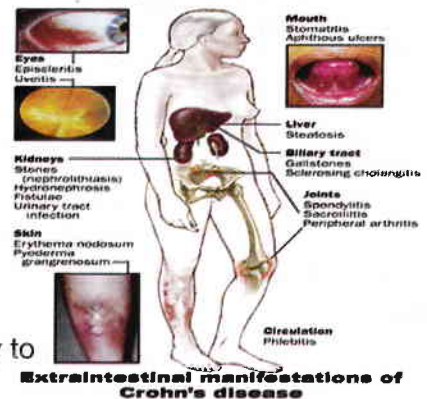
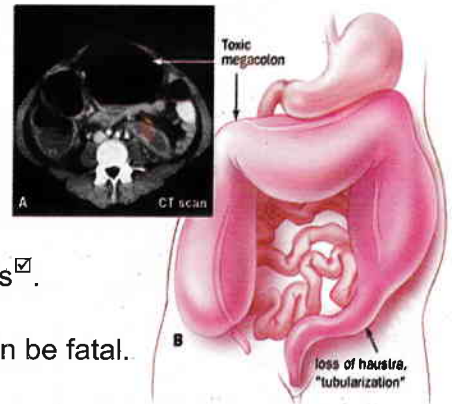
Complications

General:

1. Eye → uveitis☑.
2. Joint → spondylitis, arthritis☑ (large joint).
3. Skin → erythema nodosum, pyoderma gangrenosa☑.
4. Liver → fatty changes, cirrhosis, cholangiocarcinoma, sclerosing cholangitis☑.

Local:

1. Toxic **megacolon**☑ in (3-5%) of cases, can be fatal.
2. **Malignancy** Cancer colon☑:
 - i. The risk is higher in patients with pancolitis for > 10 years (higher risk than Crohn's disease).
 - ii. Malignancy is associated with duration as well as extent of the disease.
 - iii. Intestinal carcinoma 2ry to U.C is multicentric and more malignant than the carcinoma that occurs in previous normal person☑.
3. **Perforation** of inflamed ulcers, less commonly in U.C.
4. **Perianal suppuration** in 10% of patients 2ry to chronic diarrhea..
5. **Fibrosis** → stricture.
6. **Massive hemorrhage**☑.



Extraintestinal manifestations of Crohn's disease

- ➔ **Remission and exacerbation is a characteristic feature.** Exacerbation may be precipitated by: stress, infection, antibiotics or NSAIDs.
- ➔ **Ocular manifestations of U.C. are related to disease activity and responds to steroids and immunosuppressants except panophthalmitis.**

Investigations

1- Radiological:

Barium enema:

- Colonic shortening.
- Loss of haustrations (pipe-stem appearance☑).
- Granular mucosa☑.
- Pseudopolyps☑.

2- Instrumental:

Colonoscopy and biopsy (the best):

- Shows mucosal abnormalities.

3- Laboratory:

- **CBC:** anemia and leucocytosis in acute cases.
- **Hypoproteinemia and hypokalemia:** in severe cases.



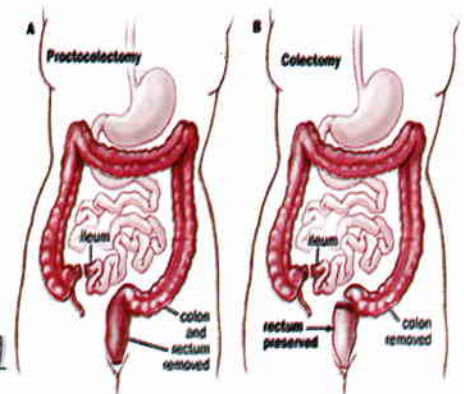
Barium Enema showing loss of haustrations on left side (pipe stem appearance)

Treatment**A- Medical treatment:**

1. Rest.
2. Diet → ↑ protein, vit. & iron, low residue diet.
3. Anti-Inflammatory drugs:
 - **Sulphasalazine** (salazopyrin): 2 – 6 gm / day according to severity.
 - **Steroids** (enema or systemic) in acute attack.
 - **Azathioprine** in severe cases.

B- Surgical Treatment: (in about 20% of cases)

- The need for surgical treatment is highest in 1st year following diagnosis.
- **Indications:**
 - 1- Failure of medical treatment.
 - 2- Rarely with complications: like hemorrhage and intestinal obstruction
- **Procedures:**
 - 1- The standard treatment is excision of the whole colon and rectum (**pan-proctocolectomy**) with permanent ileostomy (or ileo-anal anastomosis or ileal reservoir)
 - 2- The colon is excised but the rectum may be spared with ileorectal anastomosis.
 - 3- Excision of the colon and upper part of the rectum and the mucosa of the lower part of the rectum (which is the site of the disease). It is cored out leaving the muscle wall.



2- Crohn's Disease (Regional ileitis)

Definition

- Chronic transmural[□] granulomatous inflammation that can involve any area in the GIT.

Incidence

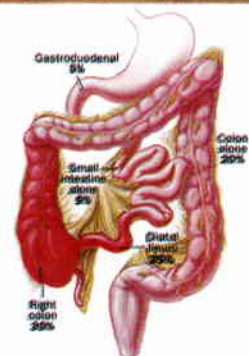
- Male = Female.
- Peak age of onset is in 2nd – 4th decades (15-30 Y).

Etiology

Unknown but the following may play a role:

- 1- Autoimmune[□] (most acceptable): in response triggered by unknown bacteria or viruses.
- 2- Hereditary factors.
- 3- Focal ischemia.

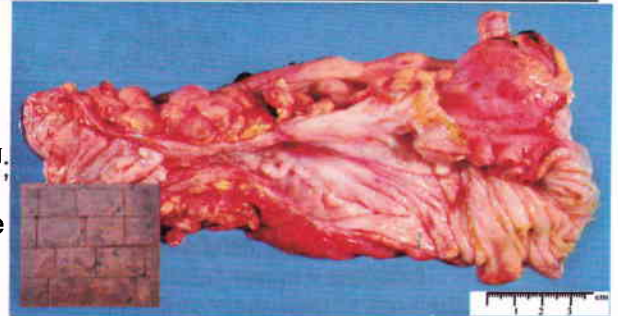
- ▶ **Etiology:** unknown, may be autoimmune
- ▶ Any part of the GIT mainly terminal ileum
- ▶ **Path.:** transmural, cobble-stone app., skip lesions
- ▶ **C/P:** - acute attacks: like acute appendicitis
- Chronic: diarrhea & rectal bleeding, wt. loss
- ▶ **Comp.:** general, local
- ▶ **Invest.:** Ba meal follow through (cobble-stone, string sign of Kantor), colonoscopy & biopsy
- ▶ **TTT:** Medical TTT if failed surgery



Pathology

Site

- The disease can affect any part of the GIT from mouth to the anal canal.
- The commonest site is terminal ileum, hence the old name "Regional ileitis".
- Next common site is the colon then the anal canal.
- Draining LNs may be affected.



Cobblestone Appearance

Macroscopic Picture

- Aphthoid ulceration is an early sign, they are often multiple, they grow & coalesce to form large areas of ulceration with intervening areas of mucosal edema, this gives "**Cobblestone appearance**".
- The affected segments are firm, thickened and heavy (hosepipe).
- There is normal bowel in between "**Skip - lesion**".

Microscopic Picture

- Early in the disease there is accumulation and attack of the base of crypts by inflammatory cells this lead to formation of aphthoid ulcers.
- **Non-caseating granuloma** infiltrating the whole thickness "transmural" from aggregations of epithelioid histiocytes surrounded by lymphocytes and Langhan's giant cells.

Clinical Picture

- Ileal disease may mimic acute appendicitis while colonic disease may present as fulminating colitis.
- **Acute attack:**
 - Picture similar to **acute appendicitis** but there is history of diarrhea.
- **Chronic cases:**
 - **Symptoms:**
 - **Diarrhea** result from mucosal inflammation, bacterial overgrowth, fistulae between bowel loops and bile salt malabsorption.
 - **Abdominal pain** is common. It is colicky, due to ↑ peristalsis induced by partial or complete obstruction.
 - **Rectal bleeding** more in colonic disease..
 - **Weight loss**, malaise, low grade fever, lethargy, anorexia, nausea and vomiting.
 - **Signs:**
 - **General:**
 - Anemia, malnutrition & aphthous ulceration of the mouth.
 - Finger clubbing, peripheral edema.
 - **Local:**
 - Usually normal.
 - Mass in the Rt. iliac fossa: tender & doughy.



Complications

- **General:**
 - Inflammatory ocular, joint affection, skin lesions and hepatobiliary lesions are similar to those of ulcerative colitis.

▪ **Local:**

- **Malabsorption** in extensive lesions or in those who had extensive resection.
- Peri-anal suppuration, anal fistula and fissure
- **Fibrosis** → stricture leading to I.O
- **Abscess and fistula** to another loop of bowel, bladder, vagina or to the skin



Death rates in Crohn's disease are 2-3 times more than general pop. especially due to malignancy

Investigations

1- Radiological:

▪ **Barium meal follow through:**

➤ **Early:**

- **Cobblestone** appearances due to longitudinal fissures, skip lesions are also seen.

➤ **Late:**

- **String sign of Kantor** (pathognomonic due to marked narrowing of the terminal ileum).

▪ **Double contrast barium enema:**

- Will show the same changes in the colon.

2- Instrumental:

▪ **Colonoscopy and biopsy:**

- Large bowel lesions prove the diagnosis.
- Biopsy of suspected anal fissure or fistula.

3- Laboratory:

- **CBC:** Anemia.
- **ESR & CRP:** both are elevated.
- **Biochemical profile and serum proteins:** low serum albumin, iron and/or folate. ↓Ca, Mg, Zn, selenium and vitamin A may occur in severe disease.



Treatment

A. Medical treatment:

- Low residue, high protein and caloric diet with supplementary vitamins and minerals.
- Antispasmodics for pain.
- Corticosteroids, sulfasalazine (Salazopyrines) and metronidazole (Flagyl®) are also given in acute cases.
- Infliximab (anti TNF) can also be used

B. Surgical Treatment:

▪ **Indications:**

- 1- Failure of medical treatment.
- 2- Presence of complications e.g. IO (the most frequent indication), chronic debilitation.
- 3- Malignant transformation.

▪ **The aim:**

- Limited resection of the affected segment to avoid malabsorption syndrome as the patient may be subjected to further resection in the future.
- If there is localized stricture → strictureplasty.

3- Bilharzial Colitis

Incidence

- The disease often affects young males in rural areas of Egypt.

Etiology

- The disease is caused by schistosoma Mansoni and less commonly by schistosoma Hematobium.

- ▶ Common in young males living in rural areas
- ▶ **Etiology:** mainly Schistosoma Mansoni
- ▶ Not precancerous
- ▶ **C/P:** Portal HTN, Dysentery
- ▶ **Invest.:** stool analysis, sigmoidoscopy
- ▶ **TTT:** Anti-bilharzial drugs + surgical intervention according to the finding

Pathology

- The usual sites are the sigmoid colon & the rectum.
- Chronic inflammatory cells surround the deposited ova leading to the formation of bilharzial granuloma.
- The disease occurs in 2 forms:
 - **Submucous form:**
 - The mucous membrane becomes hyperemic, it bleeds easily and secretes mucous.
 - In chronic cases: the mucosa develops multiple polyps and ulcerations.
 - **Diffuse form:**
 - All layers of the colon as well as the mesocolon are affected.
 - The sigmoid colon forms what is known as a "bilharzial pericolic mass" = "Bilharzioma".

The condition is not precancerous and does not produce strictures.

Complications

1. Anemia due to chronic blood loss.
2. Hypoproteinemia due to chronic protein loss.
3. Partial rectal prolapse.
4. Prolapse and strangulation of a polyp.

Clinical Picture

Symptoms

1. Clinical picture of portal hypertension (e.g. splenomegaly, esophageal varices....).
2. Diarrhea, tenesmus, and passage of mucus and blood in stools (dysentery), due to irritation by polyps (colon is wide → no I.O).
3. Weakness, dyspepsia, pallor & fingers clubbing.

Signs

1. Hard nodular mass in the left iliac fossa if pericolic mass.
2. Hepatosplenomegaly is present in many cases.
3. **DRE:**
 - Anorectal varices.
 - Multiple polyps or ulcers.

Investigations

1. Laboratory:

- **Stool analysis:**
 - Shows blood, pus and may be bilharzial ova.
- **Urine analysis:**
 - May reveal associated urinary infestations.
- **CBC:**
 - Anemia.
- **LFTs:**
 - May show hypoalbuminemia
- **Serology:**
 - COPT (Circumoval Precipitation test)

2. Radiological

- **Barium enema:**
 - Multiple rounded filling-defects (polyps)

3. Instrumental:

- **Sigmoidoscopy:**
 - Shows mucosal changes
 - Examination of fresh biopsy specimens or ulcer scrapings → shows the ova.

4. Serology:

- COPT in closed lesion.

Treatment

- **Antibilharzial drugs:** praziquantel or oxfamiquine
- **Polyyps** → polypectomy by colonoscope.
- **Ulcers** → electrocautary.
- **Excision of an affected sigmoid colon is rarely needed.**
- **If intestinal obstruction due to bilharzioma → Lt. hemicolectomy.**

Rectal Prolapse

Definition

- Abnormal descent of part or all of the upper rectum through the lower rectum and/or the anal canal to protrude outside the anus.

- **Types:** partial, complete
- **Etiology:** piles (III, IV), BPH, diarrhea
- **C/P:** swelling, discharge, comp.
- **Invest.:** anorectal manometry, EMG, cause
- **TTT:** - partial: digital reposition, Thiersch op.
- Complete: rectopexy, Thiersch op.

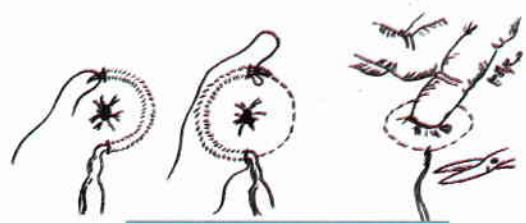
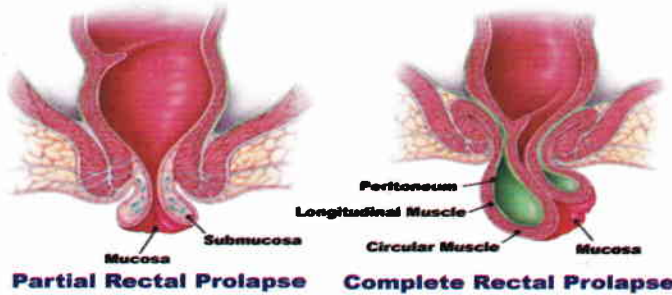
	Partial RP	Complete RP
Definition	Prolapse of rectal mucosa	Prolapse of whole thickness of the rectal wall
Etiology	1. Loss of weight (loss of supporting pararectal fat). 2. Prolonged diarrhea or whooping cough (d.t. excessive straining). 3. Advanced cases of hemorrhoids (grade III, IV). 4. BPH due to continuous straining. 5. Sphincteric atony in the elderly. 6. Iatrogenic injury of anorectal sphincter during a fistula operation.	- More common in females, while in Egypt more in young males (due to bilharzial colitis). 1. Repeated diarrhea → excessive strangulation. 2. C.T. disease (d.t. defective collagen synthesis). 3. Abnormal mobility of the mesorectum leading to lack of fixation between the rectum and sacrum. 4. In Egypt, Bilharzial proctitis and colitis → continuous tenesmus → strangulation
Pathology		
Length	< 5 cm	> 5 cm
Thickness ☒	Mucosa only	Whole rectal thickness
Complications	1- Ulceration and infection. 3- Irreducibility. 5- Fecal incontinence.	2- Bleeding. 4- Strangulation and gangrene. 6- Discharge, pruritus.

	Partial RP	Complete RP
Clinical Picture <u>Symptoms:</u> <u>Signs:</u>	1. <u>Something protruding from the anus at defecation:</u> ▪ Early: it reduces spontaneously. ▪ Late: it requires manual repositioning. 2. <u>Picture of complications.</u> 3. <u>Mucous discharge.</u> Inspection: Prolapse is best seen in squatting or lateral position and straining. Palpation: For thickness of prolapse and tone of the sphincters, Prolapse, anal sphincter, pelvic floor, PR	
Investigations DD	1. Anorectal manometer, EMG of rectal sphincters. 2. Sigmoidoscopy or barium enema: to exclude polyps, masses or any underlying cause. 1- Prolapsing hemorrhoids. 2- Prolapsing polyp. 3- Prolapsing intussusception.	
Treatment	Children 1. Correction of the cause 2. Digital reposition → if failed ↓ 3. Submucous injection of absolute alcohol or phenol in almond oil → induces fibrosis 4. Thiersch operation (perianal circlage) The anal orifice is narrowed by passing a non absorbable suture around it. The wire is tightened while the assistant's finger is inside the anus. 5. Banding or excision of redundant mucosa.	Adults: 1. Correction of the cause 2. sphincter exercises 3. Excision of the prolapsed mucosa There are various surgical procedure include: 1- Rectopexy:(laparoscopic) The rectum is mobilized and pulled up, then fixed to a mesh attached to pre-sacral (Waldeyr's) fascia and puborectalis muscle by sutures. 2- Excision of the redundant rectum , either through an abdominal or perineal approach or transanal approach called. Delorme's operation. 4. Thiersch operation N.B. continence improve after surgery in only half of the patients. 5. Perineal rectosigmoidectomy.

📌 **Digital reposition:**

- The baby should defecate on lying on his side.
- After defecation → clean the baby.
- Reduce the prolapsed mucosa
- Compress both buttocks with plaster

📌 **Rectal prolapse may be occult or manifest according to protrusion outside the anus**



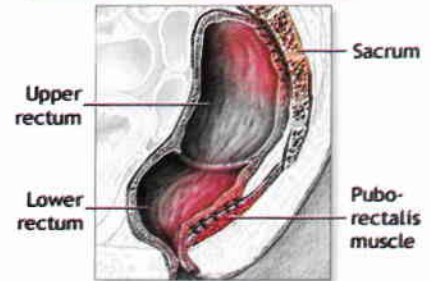
Thiersch Operation



Complete Rectal Prolapse



Partial Rectal



Rectopexy

Occult bleeding per rectum

➡ Definition

- Passage of a small quantity of blood/rectum that can't be detected by gross appearance of stools.

➡ Causes

- 1- GIT malignancy.
- 2- GERD.
- 3- Peptic ulcer.
- 4- GIT polyps

Colorectal Tumors

I- Benign Tumors

- Benign colorectal tumors usually form polyps

A- Epithelial:

1. Neoplastic:
 - Adenoma, solitary or multiple (FPC and Gardner \$)
2. Hamartomas:
 - Juvenile polyps; solitary or multiple
 - Peutz Jeghers polyps

B- Mesenchyma:

- Lipoma, Leiomyoma, or hemangioma may project in the lumen as polyp.

More Details

Epithelial:

1-Peutz Jeghers Syndrome

- Autosomal dominant[☑].
- Multiple polyps (hamartomas[☑]) in the stomach, small intestine & colon, jejunum[☑].
- Melanotic pigmentation of skin & mucous membranes[☑].
- May Present with bouts of colic & vomiting, intussusception, & BPR, anemia in childhood[☑].
- Malignant potential is small (2%)[☑].



2-Familial Polyposis Coli (FPC)

Etiology

- Autosomal dominant[☑] disease, male =female.
- Gardner's syndrome[☑] → familial polyposis associated with desmoid tumors, osteochondroma & sebaceous cysts.

- ▶ **Etiology:** AD (♂=♀), Gardner's \$
- ▶ **Path.:** affects mainly rectum & sigmoid colon
- ▶ **C/P:** at puberty, +ve family history, diarrhea, BPR, abdominal pain, IO
- ▶ **Comp.:** 100% precancerous
- ▶ **Invest.:** Ba enema, colonoscopy & biopsy
- ▶ **TTT:** panproctocolectomy + ileal reservoir

Pathology

Site

- The sigmoid colon and rectum[☑] are the commonest sites.
- They are full of multiple polyps (at least 100).

Macroscopic

- The polyps are sessile and pedunculated.

Microscopic

- Three histological types are recognized; tubular, tubulovillous and villous.



Clinical Picture

Type of patient

- Male / female at age of puberty, with +ve family history.

Symptoms

- Diarrhea, bleeding per rectum and abdominal pain.
- Intestinal obstruction due to intussusception.

Signs

- ➔ General:
 - Bad general condition.
 - Anemia.
- ➔ Local:
 - P/R → polyps.

Complications

- Carcinoma develops in 100 % of the untreated patients[□].
- The malignant potential is related to the size of the adenoma, so the highest incidence present with villous adenoma (> 2 cm).

Investigations

1- Radiological:

- **Barium enema:**
 - Multiple rounded filling defects throughout the colon and the rectum.



2- Instrumental:

- **Colonoscopy and biopsy:** Proves the nature of the disease.

3- Family screening:



Treatment

Surgical procedures are similar to those of ulcerative colitis.

- 1- **The standard treatment** is excision of the whole colon and rectum (pan-proctocolectomy[□]) with permanent ileostomy (or ileo-anal anastomosis or ileal reservoir)
- 2- **Less popular procedures:**
 - a) The colon is excised but the rectum may be spared with ileorectal anastomosis.
 - b) Excision of the colon and upper part of the rectum and the mucosa of the lower part of the rectum (which is the site of the disease is cored out leaving the muscle wall).

DD

	Familial polypi	Bilharzial polypi
Age	Young age	Adult
Sex	Both are equal	More in males
F.H	+ve	-ve
Site	Mainly sigmoid, rectum	Mainly sigmoid, rectum
Micro	Adenomatous polypi	Bilharzial granuloma, +ve ova
Danger	Precancerous 100 %	-ve

Carcinoma of the Colon and Rectum

Definition

- Cancer colon is defined intra-operatively to be proximal to the point where the 2 anti-mesenteric taenia coli converge.
- Cancer rectum is distal to this point.

Incidence (epidemiology)

- One of the leading causes of death in the western countries.
- **Age:** - Usually above 50 years but not rare before this age. Peak incidence is at 60s.
 - Incidence increases if there is a first degree relative with cancer colon and may occur at earlier age.
- **Sex:** - Cancer cecum more common in females
 - Otherwise → more common in males

- ▶ **PDF:** chronic irritation, precancerous lesion & genetic factors
- ▶ Usually affect rectum and sigmoid colon
- ▶ **C/P:** ♂ > 40 y. with progressive constipation
With or without obstruction
- ▶ **Comp.:** IO (20%), perforation, fistula, bleeding
- ▶ **Invest.:** - with IO: colonoscopy and biopsy, Ba enema, spiral CT
- without IO: X-ray
- ▶ **TTT:** - Without IO: operable or non-operable
- With IO: R&M, then acc. to general condition

Etiology and predisposing factors

A. CHRONIC IRRITATION:

1. Dietary factors: low fiber and high animal protein → constipation → increase transit time → production of fecaptin → carcinogenic.
2. Smoking, alcohol.
3. Uretero-colic anastomosis.
4. IBD (ulcerative colitis) → if pan-colitis > 10 years.

B. BENIGN PRECANCEROUS LESIONS:

- Colonic polyps especially villous adenoma more than 2 cm.

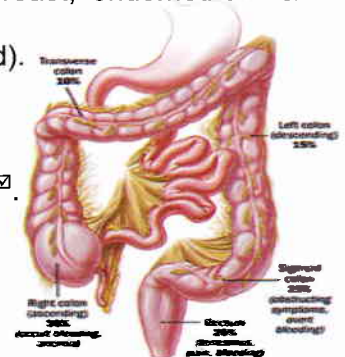
C. GENETIC FACTORS:

1. FPC and Gardner's syndrome → 100% precancerous by the 5th decade, more on Lt. side.
2. **Lynch syndrome:**
 - Cancer colon, more on Rt side, & carries better prognosis than familial polyposis induced cancer.
 - Type I → cancer colon.
 - Type II → cancer colon ± endometrial carcinoma ± cancer ovary.
3. **Le Fraumini syndrome:**
 - Defective P53 gene (suppressor gene) → cancer breast, endometrium & colorectal carcinoma.
4. Mutations in **APC gene** and **K-RAS gene** (recently discovered).

Pathology

Site

- 2/3 of cases are situated in the rectum and sigmoid colon[□].
- 25 % in the caecum and the right colon (10% in cecum).
- The remainder is distributed over the rest of the colon.
- Transverse colon is the least common affected site.
- Multiple tumors in 5% of cases.

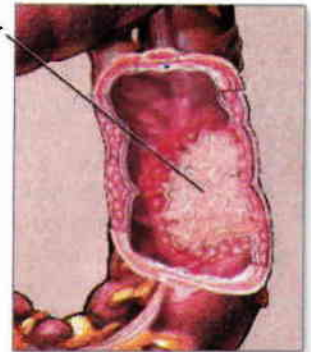


Macroscopic Picture

- Annular infiltration → causes obstruction
- Tubular infiltration
- Ulcerative → most common, especially in the left side.
- Cauliflower → least malignant, usually in the right side.

Microscopic Picture

- Adenocarcinoma.
- Colloid carcinoma.
- Anaplastic carcinoma.
- Malignant melanoma. (esp. in the rectum)
- Carcinoid, lymphoma, GIST.



Spread

1- Direct spread:

(Mainly in upward direction)

- First in the colonic wall itself ($\frac{1}{4}$ circle every 6 months)
- Then to the neighboring organs → fistula formation (especially ulcerative type).
- Fascia of Denonvillier: strong fascia lying in front of the rectum retards the spread of rectal cancer to the bladder and prostate.

2- Lymphatic spread:

- In the right and transverse colon: into epicolic, paracolic and then into superior mesenteric L.Ns.
- In left colon: into epicolic, paracolic and then into inferior mesenteric L.Ns
- Nodal involvement is found in 40% of case coming to operation.

3- Blood spread:

- mainly to the liver (hepatic metastases is found in 20% of cases coming to surgery).

4- Transperitoneal spread:

- Leads to peritoneal nodules and ascites.

Duke's pathological staging

Stage A

(Of prognostic value) ☑

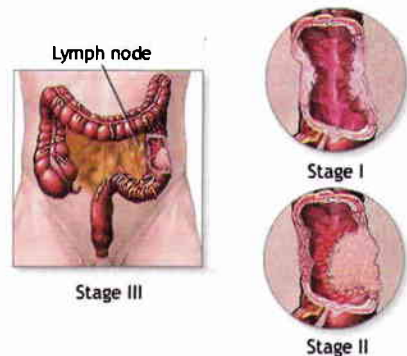
- Limited to the bowel wall & not extending beyond muscularis propria.

Stage B

- Extends outside the bowel wall to subserosa, serosa and nearby tissues without nodal involvement.

Stage C

- There are deposits in the regional LN's.
 - C1: extends to pararectal or paracolic LNs.
 - C2: extends to LNs accompanying the supplying BVs.



Staging of Colorectal Carcinoma

NB.

- **Modified Duke's staging** ☑: (not described by Duke).
- There is D stage: distant metastasis.

TNM staging

Tx: primary tumor cannot be Assessed	Nx: nodal metastasis cannot be assessed.	Mx: distant metastasis cannot be assessed.
T0: no primary tumor identified	N0: no nodal metastasis.	M0: no distant metastasis.
Tis: carcinoma in situ	N1: 1-2 pericolic\perirectal nodes involved.	M1: distant metastasis.
T1: tumor invades into submucosa but not penetrating musculosa.	N2: 3 or more nodes.	
T2: invasion of musculosa.		
T3: invades to the subserosa, but not reaching the serosa.		
T4: invades serosa or other organs.		

- TNM surgical staging exists and is even of more important nowadays.

Complications

- 1- **IO occurs in 20 % of cases esp . with left colon tumors** this is due to
 - The smaller lumen of the Lt. colon.
 - Stools tend to be more solid.
 - Carcinoma tends to be of the stenosing variety.
 - It may be the first presentation.
- 2- **Perforation or the formation of enterocolic or vesico-colic fistula**
- 3- **Fistula formation (malignant fistula)**, either with bladder or vagina.
- 4- **Bleeding, chronic bleeding is the rule while massive bleeding is rare.**
- 5- **Complications due to spread e.g: ascites, jaundice.....**

Clinical Features

Any ♂ > 40 yrs with progressive constipation > 2wks → Cancer colon

(Depends on the site and the presence of obstruction)

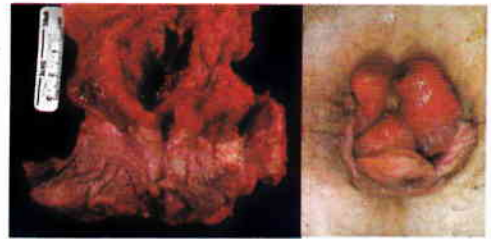
A. WITHOUT OBSTRUCTION**■ Lt. colon cancer**

1. Change of bowel habits (**it is the most important presentation**):
 - Usually progressive constipation or constipation alternating with diarrhea
 - **Spurious diarrhea** → passage of mucous & blood in the early morning may be present (especially with the colloid variety).
2. Bleeding per rectum:
 - Fresh, but not massive bleeding.
3. Symptoms of intestinal obstruction (especially with sigmoid cancer):
 - Either acute, subacute or chronic.
 - Progressive constipation, abdominal distension & colicky lower abdominal pain.
4. Mass on the Lt. side of abdomen:
 - Rarely presented by a mass .
 - If palpable mass → due to impacted stools above obstruction



Rectum and sigmoid colon

1. Bleeding per rectum, usually slight; rarely massive.
2. Tenesmus (sense of incomplete evacuation).
3. Painless unless spread outside the wall (to sacral plexus, neighboring organs).
4. Bladder symptoms (especially if colovesical fistula).
5. Sometimes, may be presented by piles (bleeding/rectum)
6. DRE → palpable if within 10 cm from the anal verge.



Transverse colon

✓ (vague clinical picture)

- Dyspepsia, anemia, epigastric mass.
- May be mistaken for cancer stomach.

D.D. of mass in the epigastrium:

1. Cancer stomach.
2. Cancer head of pancreas.
3. Hepatoma of the Lt. lobe.
4. Aortic aneurysm.

Caecum and ascending colon

1. Unexplained loss of weight & appetite.
2. Iron deficiency anemia (due to chronic bleeding).
3. Hard mass in the right side of abdomen.
 - Differentiated from appendicular mass by long duration and absence of toxemia and tenderness.
4. Recurrent attacks of pain in the right iliac fossa.



DD: appendicular mass → toxemia, tenderness, relatively short duration

Whenever rectal bleeding occurs in a middle aged or older individual even in the presence of piles → co-existing rectal cancer should be excluded.

B. WITH OBSTRUCTION

- Commonest with left sided cancers, rare with other types.
- In the left side → gradually progressive constipation, distension and colicky lower abdominal pain.
- In the right side:
 - May be the apex of intussusception → intermittent obstruction.
 - May obstruct the ileocaecal valve and terminal ileum.

D.D.

- Massive bleeding/rectum (See D.D. book).

Investigations

1. For diagnosis:

A. Without obstruction:

1. **ENDOSCOPY** (sigmoidoscopy if < 60cm from anus, colonoscopy if more) reaching up to caecum:

- Now it's the investigation of choice for old patients with altered bowel habits or rectal bleeding.
- Mandatory in every patient above 40 years with complaint of piles.
- Advantages:
 - ✓ Taking a biopsy (which is mandatory).
 - ✓ Can detect multicentric lesions (7%).



2. **BARIUM ENEMA** (better a double contrast barium enema):

- The cauliflower tumor appears as a fixed irregular filling defect.
- Annular strictures of the left colon show a characteristic "apple core appearance"☐.
- Complete arrest of dye in severe stricture or complete obstruction.



3. **SPIRAL CT:**

- Done if the above measures are contraindicated.
- In some centers, it is the standard for patients > 80 years old.

B. With obstruction:

1. Plain X-Ray abdomen, erect (multiple fluid levels) and supine.
2. Electrolytes and urea.

2. For staging:

- **Endoluminal (trans-rectal) U/S** → detects extent of local infiltration.
- **Abdominal U/S and CT scan** → metastasis to LNs, liver and abdomen.
- **Chest X-Ray or CT scan**
- **Bone scan**
- **IVU** → possibility of involvement of ureters.
- **PET scanning** → malignant nodules elsewhere.

3. For preoperative preparation:

- **CBC** (iron def. anemia, coagulation profile) – **KFTs** – **LFTs** – **FBS** – **ECG**

4. For follow up:

- **Tumour Markers**☐: Carcino Embryonic Antigen (CEA) → it is not specific & is of prognostic rather than diagnostic value, detect recurrent cancer colon☐.

Treatment

A. WITHOUT OBSTRUCTION

I. Operable cases (curable):

1. Preoperative preparation:

- Dietary restriction to only fluids 48 hours before surgery.
- Purging the colon on the day of surgery + rectal enemas.
- Prophylaxis against postoperative DVT (heparin + elastic stocking).
- Prophylactic antibiotics (Neomycin, Flagyl)

2. Elective radical resection:**- Idea:**

- a) Intra-operative assessment of operability and metastasis.
- b) Removal of tumor-bearing segment with its lymphatic drainage in one mass + restoring bowel continuity.
- c) Removal of the LNs requires ligation and division of the supplying vessels at their origin (since the LNs lie close to them). This necessitates removal of the whole part of the colon supplied by the divided vessels.

- Operations (according to the site):**a) Tumors of the caecum:**

- Rt. hemicolectomy (with division of ileocolic and Rt. colic vessels at their origin + end-to-end ileo-transverse anastomosis)

b) Tumors of the ascending colon or hepatic flexure:

- Extended Rt. hemicolectomy (with division of Rt. branch of middle colic vessel in addition to the above).

c) Tumors of the transverse colon:

- Transverse colectomy (with division of middle colic vessels at their origin + end-to-end anastomosis).

d) Tumors of the splenic flexure or descending colon:

- Lt. hemicolectomy (with division of inferior mesenteric vessels at their origin + end-to-end anastomosis).

e) Tumors of the sigmoid colon:

- Sigmoid colectomy (with division of sigmoid vessels at their origin + end-to-end anastomosis).

f) Tumors of the rectum:**1. Tumors of the upper third of the rectum:**

- Treated by anterior restorative resection where the IMA is divided (or sometimes the left colic is spared) and the sigmoid colon, and upper half of the rectum are excised.
- Bowel continuity is then restored.

2. Tumors of the lower third of the rectum:

- Treated by an abdominoperineal resection (of Miles).
- It is similar to the above, but the whole rectum is excised together with anal canal, and a terminal colostomy is fashioned.

3. Tumors of the middle third of the rectum:

- Used to be treated by abdominoperineal resection and a colostomy like lower third tumors.
- Two factors made a change in this policy towards anterior restorative resection:
- A 2 cm safety margin in the rectum below the tumor is compatible with radicality, thus leaving enough distal rectum for anastomosis.
- The development of circular staplers facilitated this technically difficult procedure.

g) Liver metastasis:

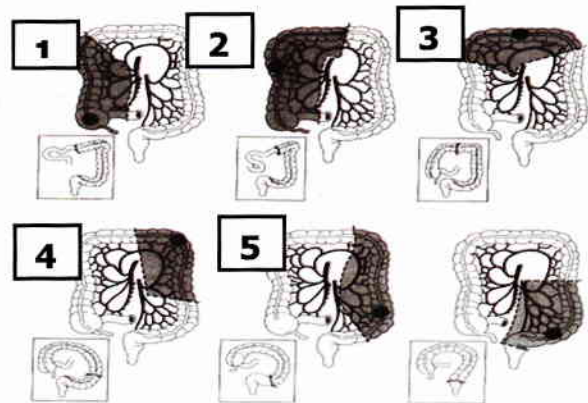
- Resection if <4, limited to one lobe and no other metastasis.

II. Inoperable cases:

- Resectable: resection is preferred as it is adenocarcinoma (insensitive to radiotherapy or chemotherapy)
 - Irresectable: operation is done to avoid obstruction
 - Right colon → side-to-side ileo-transverse anastomosis.
 - Left colon and rectum → proximal colostomy.
- Tumors of the rectum show partial response to local radiotherapy and cancer colon shows low sensitivity to chemotherapy (5-fluorouracil).
- Chemotherapy is preferred to radiotherapy, d.t. its disadvantages:
- Hemorrhage.
 - Infection.
 - Ulceration.

B. WITH OBSTRUCTION:

- Resuscitation and monitoring, then:
- According to general condition:
 - If good general condition → exploration
 - Operable → radical resection according to site.
 - Inoperable → surgery to avoid obstruction later.
 - If bad general condition (unfit for surgery) → colostomy → improve general condition → colon resection.

**1- Rt. Hemicolectomy.****2- Extended Rt. Hemicolectomy.****3- Transverse colectomy.****4- Lt. hemicolectomy.****5- Sigmoid colectomy.****Prognosis**

- Worse prognosis of the colorectal cancer is encountered in the following conditions:
1. Young age at clinical presentation.
 2. Colonic obstruction or perforation.
 3. Blood vessel or lymphatic obstruction.
 4. Liver and/or distant metastases.
 5. Aneuploidy.

More Operative Details

Rt. Hemicolectomy**1- Removal of:**

- The terminal 10 cm of ileum.
- Caecum.
- Ascending colon.
- Hepatic flexure.
- Rt. ½ of transverse colon.
- Peritoneum of post. Abdominal wall between the resected colon and sub mucous vessels as it contains lymph vessels & LNs.

2- Ileo-transverse anastomosis.

Extended Rt. Hemicolectomy

- Rt. Hemicolectomy + removal of Rt. 2/3 of the transverse colon.

Transverse Colectomy**1- Removal of:**

- Transverse colon and mesocolon.
- 2 flexures.
- Greater omentum.

2- End to end anastomosis.**Lt. Hemicolectomy****1- Removal of:**

- Lt. 1/3 of transverse colon.
- Splenic flexure.
- Descending colon.
- Wedge of peritoneum of post. Abdominal wall.

2- End to end anastomosis**Sigmoid Colectomy****1- Removal of sigmoid colon & mesocolon.****2- End to end anastomosis.**

Colostomy

➡ See operative book

Section D

The Large Intestine

Anal Canal



Imperforate Anus

Incidence

- 1 / 5000

Types

- ▶ **2 types:** **A.** High anomaly (agenesis, atresia)
B. Low anomaly (covered, membranous anus)
- ▶ **C/P:** - **General:** to exclude **VACTERL**
 - **Abdominal:** IO may be present
 - **Local:** meconium at tip of penis → high anomaly
Impulse on crying at the anus → low anomaly
- ▶ **Invest.:** invertogram 24 hrs after birth, IVP, urine analysis
- ▶ **TTT:** **a.** High anomaly: staged surgery (3 stages)
b. Low anomaly: Only perineal surgery

1. High anomalies (More common)

- Failure of the rectum to pass through the pelvic floor (proximal to the pelvic floor).
- Usually associated with fistulous communication with posterior urethra in males or vagina in females.
- In high anomalies (M : F = 2 : 1)

Types:

a- Anorectal Agenesis:

- Rectum:**
 - Forms a blind pouch above the pelvic floor with fistulous communication with UB, urethra or vagina.
- Anal canal:**
 - Is absent.

➤ Persistent Cloaca:

- Only in females.
- The bowel, UB and genital tract are all opened at a common wide cavity (Cloaca)

b- Rectal Atresia:

- Rectum:**
 - Forms a blind pouch above the pelvic floor.
- Anal canal:**
 - Forms a blind pouch below the pelvic floor.

2. Low anomalies (Less common)

- Distal to the pelvic floor.
- In low anomalies (M : F = 1 : 2)
- Not associated with other anomalies
- No defect in anal sphincter

a- Covered Anus:

The anal canal is covered by skin bar and usually opens into an ectopic site anterior to normal position.

b- Membranous Anus:

There is a membrane at the dentate line → retained meconium → bulging of this membrane.

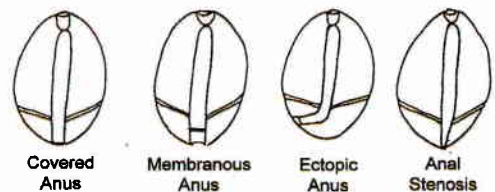
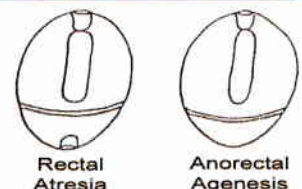
Associated Anomalies

VACTERL, more common in high than low anomalies.

Clinical Picture

General examination

To exclude associated congenital anomalies.



Anal Stenosis

Abdominal examination

There may be evidence of intestinal obstruction.

Examination of the perineum

1. Presence of anus, its size and site
2. Presence of an anal dimple
3. **If there is an impulse on crying at the site of the anus → low anomaly.**
4. Subcutaneous fistulous track full of meconium → ectopic anus
5. **Meconium at the tip of the penis → fistula to the bladder or urethra.**
6. If the thermometer can be introduced into the anus for > 1 cm, this excludes rectal atresia.

DD

(Of neonatal intestinal obstruction[□]):

1. **Duodenal atresia:**
→ Vomiting (bile stained or not) → Double bubble sign on x-ray.
2. **Volvulus neonatorum:**
→ Abdominal colic, bile stained vomiting & bleeding per rectum.
→ Doppler shows affected bowel blood flow.
3. **Meconium ileus:**
→ Thick meconium
→ Gastrografen is diagnostic & therapeutic.
4. **Hirschsprung disease (long segment):**
→ Barium enema shows stenotic colon. → Biopsy is diagnostic.

Investigations

1. Radiological:

- **Plain X-ray (invertogram):**
 - 24 hours after birth[□] (enough time for gas to travel along the colon to reach the distal end of the bowel), the infant is held upside down for a few minutes with radio-opaque marker (e.g. coin) on the possible site of the anus.
 - We can differentiate between high and low anomalies by:
 1. Measuring the distance between coin and distal gas-shadow:
 - ⇒ If < 1 cm → low anomaly.
 - ⇒ If > 1 cm → high anomaly.
 2. Determining the site of termination of bowel:
 - ⇒ Proximal to the pubo-sacral line → High anomaly
 - ⇒ Distal to pubo-sacral line → low anomaly
- **IVP:** (In high anomalies)
 - For urinary anomalies
 - If bladder gases are present → high with fistula.



2. Laboratory:

- **Urine analysis:**
 - If meconium is present → fistulous communication with UB

- **Associated anomalies must be excluded:** e.g.
 - Esophageal atresia → catheter test
 - Cardiac anomalies → echo.
 - Renal anomalies → abdominal U/S. IVP.

Treatment

1. High anomalies:

- ⇒ Treated with staged surgery:
 - First stage: Temporary colostomy
 - Second stage: Anorectal pull-through (abdominal approach)
 - Third stage: closure of colostomy.

Results are not satisfactory because continence may be weak.

2. Low anomalies:

- Only perineal surgery.

3. Treatment of associated anomalies if found.



Pilonidal Sinus

- It is a condition related to skin overlying the sacrum not an anal disease, but mentioned here because of its closeness and possible misdiagnosis as an anal fistula.

Definition

- Subcutaneous granulation cavity described as a nest (latin-nidus) containing hair (latin-pilus) and connected to the skin by midline openings (latin-sinus)

- ▶ **Def.:** it is a disease of the skin covering the sacrum not an anal canal disease
- ▶ **Etiology:** unknown (cong. & acquired theories)
- ▶ Commonly in the upper part of the anal cleft
- ▶ **C/P:** young adult ♂, may be asymptomatic, Discharge☑, Pain if abscess is formed
- ▶ **TTT :** A- Abscess: AAA + drainage
B- Sinus: Laying out (or localized excision) of the cavity and side tracks

Incidence

- More in young adult males☑ with dark dense strong hair☑.

Etiology

- The actual cause is unknown☑, possible theories are:

1- Congenital theory

- Infection of congenital dermal appendages overlying the lower sacral region that presents after puberty☑.

2- Acquired theories

- A- Jeep bottom theory☑: prolonged sitting on hard seats changes the direction of the hairs.
- B- Loose hairs from head or back fall on the skin over the sacrum and coccyx, as a result of pressure from sitting, rolling movement of the buttocks, sweat and poor hygiene; the hairs are drawn through the skin to accumulate in subcutaneous tissue.

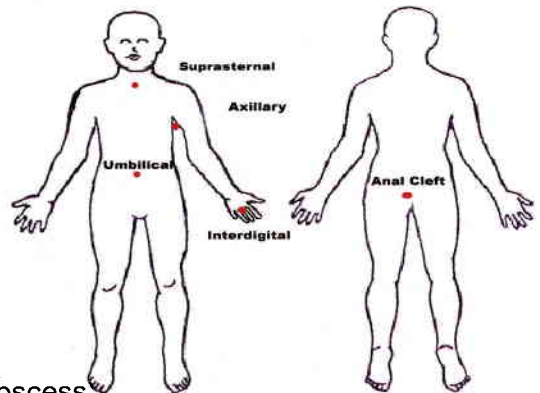
This is favored by:

- a- The tip of the hairs is always directed inwards
- b- There are only loose hairs in the cavity without hair follicle.
- c- The condition may occur in other sites.
- d- Liability to recur

Pathology

Sites

- 2- Upper part of anal cleft (most common ☑).
- 3- Others:
 - Axilla, umbilicus, Interdigital (in barbers), suprasternal notch.



Microscopically

- Subcutaneous granulation tissue cavity containing loose hairs, the cavity opens on the skin in the middle line by one or more openings.
- Aerobes and anaerobes proliferate causing an abscess.

Clinical Picture

Symptoms

- 1- May be asymptomatic.
- 2- Local discomfort, **discharge** ☑ (the most important, characteristically foul containing hair).
- 3- Painful swelling if abscess is formed.

Signs

- 1- Discharging midline and lateral openings with loose hairs protruding from it.
- 2- If abscess if formed:
 - Throbbing pain.
 - Redness, tenderness.
 - Pus oozing from primary sinus or open laterally or inferiorly producing secondary sinuses.



DD

- 1- Perianal abscess.
- 2- Anal fistula.

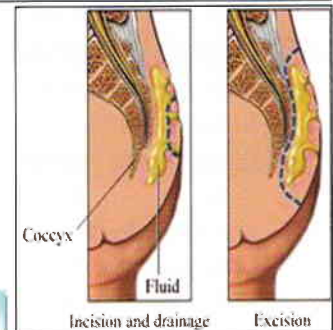
	Pilonidal sinus	Peri-anal fistula
Site	near mid-line over coccyx	Close to anal verge
Probing	Upward & forward towards sacrum	Towards anal canal.

Treatment

Pilonidal Abscess

- Rest, antibiotics, antipyretics, anti-inflammatory.
- Abscess is drained.
- Removal of loose hairs.
- The wound is left open to heal with granulation tissue.
- If a sinus recurs → treated by elective surgery.

Reccurence ☑ after treatment is the main challenge



Pilonidal Sinus

- Sinuses are identified by methylene blue or hydrogen peroxide.
- **Different options are available:**
 1. **Laying out of the cavity and side tracks and curettage:**
 - Phenol cauterization may be applied to the tracks.
 - The resulting wound is left open, packed and is allowed to heal by 2ry intention.
 2. **Localized excision of the cavity and side tracks:**
 - The wound may be left open to granulate or is closed by sutures to accelerate healing.
 3. Wide excision of the skin and subcutaneous tissue down to periosteum of the sacrum should no longer be practiced.

Anal Fissure

Definition

- An **elongated ulcer** which occurs in the **long axis** of the **lower anal canal**.

Incidence

- **Sex:** males and females are equally affected.
- **Age:**
 - Most common in middle age☑.
 - Not common in elderly.
 - Not rare in children.

Etiology

A. No definitive cause: is found in the **majority**, postulated mechanisms are:

- ▶ **Trauma:**
 - Hard stool (in constipated patients) → injury to the least supported site, i.e., *midline posterior fissure*.
 - Repeated deliveries → damage of perineal body → loss of ant. Anal support → *midline anterior fissure*.
- ▶ **Ischemia:**

May play a role in development of the condition.
Midline posterior fissure☑ is the most common as it is **least vascular**.

B. Definitive cause: is found in a **minority**.

- **IBD especially Crohn's disease**☑.
- **STDs.**
- **Iatrogenic.**
 - Large enema, endoscope.
 - Post hemorrhoidectomy: removal of too much skin → stenosis → injury of mucosa by hard stools.

These fissures may be *at any site* & even multiple

- ▶ **Inc.:** more in middle age, ♂=♀, midline post.
- ▶ **Etiology:** trauma by hard stool (constipation), ischemia, IBD, STDs, iatrogenic
- ▶ **C/P: A- Symptoms:**

Pain, constipation, bleeding
- ▶ **B- Signs:**
 - **Acute fissure:** Anal verge is contracted, DRE: is better to be avoided
 - **Chronic fissure:** sentinel pile
DRE: button hole induration
- ▶ **Invest.:** clinical diagnosis + for the cause
- ▶ **TTT: a. Acute**

Lifestyle changes + Medical TTT & if failed lateral sphincterotomy
- ▶ **b. Chronic**

Lateral sphincterotomy + Fissurectomy

Pathogenesis

It is a vicious circle

(NB. Pain is the main pathogenic factor in acute fissure)

Pathology

Site:

- Usually in the midline.
 - Midline posteriorly (90%)
 - Midline anteriorly (10%): Especially common in multiparous females.
 - Anterior and posterior fissures may coexist.
- May be anywhere as in Crohn's disease.

Pathological changes:

Acute Fissure

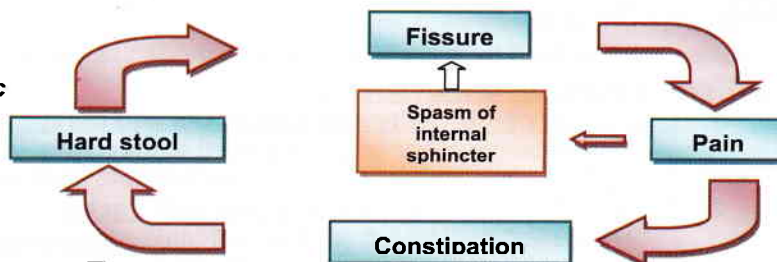
- Superficial tear present in the sensitive part (below dentate line) → pain.
- Pain → spasm of internal sphincter → ↓ blood flow → prevents healing of fissure.

Chronic Fissure

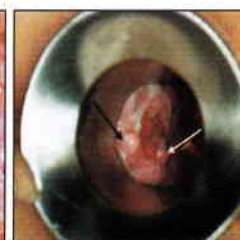
- If acute fissure is not treated well, it will proceed to chronic stage with secondary pathological changes:
 - a- Margins: become indurated, thick and fibrotic.
 - b- At the upper end of fissure: an anal papilla may develop.
 - c- At the lower end of fissure: sentinel pile (skin tag) may develop.

In neglected cases

- Stenosis of anal canal due to fibrosis of internal sphincter.
- Anal suppurum may develop → perianal abscess.



Acute Fissure



Chronic Fissure

Clinical Picture

- Usually following attack of constipation with hard stools.

Symptoms

1. Pain:

- Is the main symptom.
- Character: sharp, agonizing.
- Site: localized to the anus, can radiate to the coccyx or to the genitalia.
- Starts: at defecation.
- Lasts: for about 1 hour after defecation, and ends suddenly.
- Course: may have remissions for days or weeks.

- In chronic cases the pain is milder but more persistent and bleeding is less

2. Constipation:

- The severe pain forces the patient to postpone defecation → more constipation → ↑ fissure (vicious circle).

3. Bleeding:

- Only a slight streak of blood (bright red) on the surface of the stool.

4. Slight anal discharge and pruritis:

- If an abscess forms and bursts, there will be purulent discharge.

5. Reflex symptoms:

- Burning micturition, dysmenorrhea and pain along the thighs.

Signs

➤ In acute anal fissure:

- Inspection:
 - The anal verge is tightly contracted, puckered anus.
 - If the 2 gluteal folds are gently pulled laterally, a small tear will be seen.
- DRE:
 - Better to be avoided[☐], as it is very painful.
 - If it is essential to exclude other pathology, it should be done under general anesthesia.

➤ In chronic fissure:

- Inspection:
 - The fissure can be seen.
 - An anal papilla or a sentinel pile may be present.
- DRE:
 - Fissure is fibrotic & indurated (*Button hole induration*).
 - Sphincter is fibrosed.

DD

Of painful anal conditions

1. Anal fissure.
2. Perianal suppuration.
3. Prolapsed strangulated piles.
4. Acute perianal hematoma.
5. Carcinoma of the anus.
6. Proctalgia fugax (idiopathic).
7. Crohn's disease

Complications

1- Fissure abscess → fistula.

2- Acquired megacolon.

Investigations

It is a clinical diagnosis, however, if multiple and in uncommon sites

→ Search for a specific cause e.g. Crohn's disease → biopsy

Preoperative Investigations: CBC , LFTs,

Treatment

I. Acute Fissure (Essentially medical, but sometimes surgery is needed)

❖ Life style changes:

- High fiber diet (fruits, vegetables & cereals).
Aim: to make stool bulky & avoid constipation
- Laxatives (e.g. lactulose syrup)
Aim: to soften the stool
- Sitting in a warm bath after using the toilet.
Aim: relieve the spasm & pain

❖ **Medical treatment:**

- Chemical sphincterotomy by:
 - ⇒ Glyceryl trinitrate (GTN) ointment (2-10%):
 - Mechanism: relaxes the sphincter and improves the blood flow.
 - Success rate: about 60-70%
 - Side effects: headache, recurrence after stopping treatment.
 - ⇒ alternatives:
 - Ca channel blockers (diltiazem, nifedipine)
 - local injection of botulinum toxins
- A local anesthetic ointment is introduced gently as 2 % or 5% lignocaine.
- Local steroids (hydrocortisone) to relieve pain & inflammation.

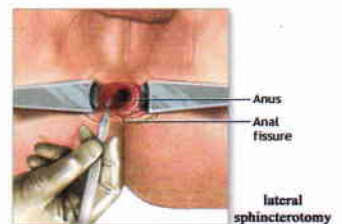
- **Aim of medical tt** is to break the vicious circle
 - To relieve pain, relax sphincters & improve the blood supply to promote healing
- **Advantage of medical treatment:**
 - No risk of incontinence.
 - Complete healing can be expected in 50% of patients with acute fissures.

❖ **Surgical treatment:**

- **Lateral sphincterotomy**☑:
- Indicated if all previous measures fail to relieve pain within a few days.

II. Chronic Fissure➔ **Treatment is surgical:**

- ⇒ **If the fissure is not very chronic:**
 - A lateral internal sphincterotomy operation is very successful.
 - The internal sphincter is divided at the 3 o'clock position. The operation is done by the closed or open method.
- ⇒ If the fissure is very fibrosed with a sentinel pile:
 - The best procedure is to do fissurectomy and posterior internal sphincterotomy.
 - A triangular segment with its apex upwards and including the fissure, anal papilla and sentinel pile is excised.
 - The excision includes the fibrosed part of the internal sphincter muscle as well.



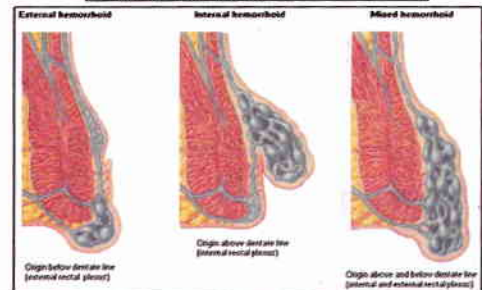
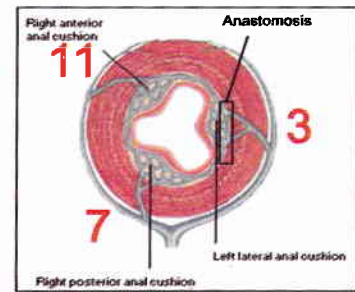
Hemorrhoids (Piles)

Introduction

- **In Greek:** hema = means blood, while rhoos = mean flowing
- **In latin:** Pila = means a ball.
- Therefore, the accurate terminology should be hemorrhoids if the main complaint is bleeding per rectum or piles if the main complaint is prolapse.

Anatomy

Normally, the terminal branches of superior rectal vessels (portal) form vascular plexus with middle and inferior rectal vessels (systemic) beneath the epithelium lining of anal canal called (anal cushions) which are arranged at the 3, 7, 11 O'clock position around the circumference of the anal canal, so any congestion, enlargement and prolapse of these A – V cushions constitute piles.



Types

- A- Internal piles = above dentate line and covered by mucosa.
- B- External piles = below dentate lined and covered by skin.
- C- Intero-external piles (the most common).

A- Internal Piles

Definition

- Dilated tortuous superior hemorrhoidal plexus of veins.

Etiology

Primary hemorrhoids (most common)

Predisposing factors:

- Hereditary factors (congenital weak mesenchyme)
 - a- Leading to weakness of venous walls or abnormally large supplying arteries.
 - b- Evidence:
 - +ve family history.
 - Varicose veins of the lower limb usually accompany it.

- ▶ **Etiology:** - Mostly 1ry: PDF (congenital weak mesenchyme), exacerbating factors
- 2ry: pregnancy, rectal carcinoma, P.H.
- ▶ **C/P:** Bleeding, prolapse, pruritus, pain
- ▶ **Comp.:** due to bleeding or prolapse
- ▶ **Invest.:** clinical diagnosis + cause (if 2ry)
- ▶ **TTT:** **A. Primary :**
 - ① 1st and 2nd degree: - Conservative TTT
 - Injection sclerotherapy
 - Rubber band ligation
 - ② 3rd and 4th degree: Hemorrhoidectomy
- B. Secondary:** TTT of the cause
- TTT of complications if present

- **Morphological factors:**
 - I.e. weight of blood column unassisted by valves → high venous pressure in the rectum.
 - Evidence: in quadrupeds, gravity aids venous return from the rectum, so hemorrhoids are very rare in animals.
- **Anatomical factors:** superior rectal vein radicals.
 - a- Lie unsupported in the loose submucous connective tissue → may prolapse
 - b- Penetrate muscular layer → may be compressed by muscle contraction e.g. during straining.
 - c- Have no valves → high venous pressure

Exacerbating factors:

- Straining (e.g. with constipation): very potent cause
- Diarrhea & dysentery: less important cause.

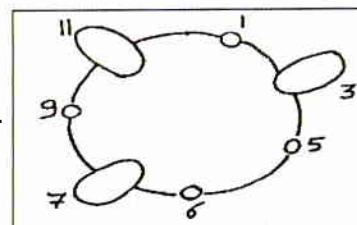
Secondary hemorrhoids

- **May occur with:**
 1. **Pregnancy:**
 - Due to raised intra-abdominal pressure and the relaxing effect of progesterone.
 - If piles persist after pregnancy, it is considered primary piles.
 2. **Carcinoma of the rectum:**
 - May obstruct the tributaries of the superior rectal vein, so it should be excluded in cases of bleeding per rectum "specially in old age".
 3. **Portal hypertension:**
 - It leads to anorectal varices.

Pathology

Types & sites

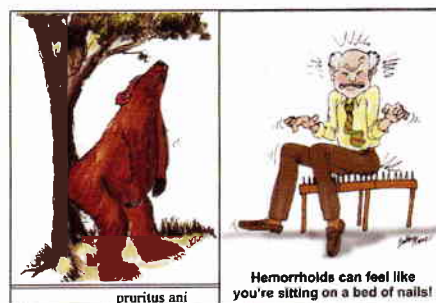
- **Mother piles:**
 - Main tributaries of superior rectal vein.
 - Present at 3, 7, 11 o'clock in lithotomy position.
- **Daughter piles:**
 - Branches of mother piles.
 - Present in between mother piles.



Clinical Picture

Symptoms

1. **Bleeding per rectum:**
 - The most common ☒ and earliest complaint.
 - Bleeding is characterized by:
 - Painless ☒, occurs at the end of defecation, **bright red**, few drops that are separate from stools.
2. **Prolapse:**
 - The vascular cushions enlarge and start to descend below the dentate line.
 - Early there is heaviness, then according to clinical degree.
3. **Pruritus and anal discharge**
 - Patients, who have prolapse of their piles beyond the anal verge, will complain of mucous discharge, which may cause pruritus ani.



4. **Pain:** only in complicated cases[□].
5. incontinence.

Signs

1. **Inspection:**
 - Prolapsed piles (4th degree).
 - Unprolapsed piles may come into view with straining.
2. **DRE:**
 - Internal piles cannot be felt (compressible) unless thrombosed.
 - It is essential to exclude cancer rectum.



It is very important to exclude[□]:

- a- Secondary causes.
- b- Other manifestations of congenital mesenchymal weakness.

Uncomplicated piles are not painful

Clinical Degrees of Piles According to Prolapse

First degree

- The patient has **only bleeding** but no prolapse of piles, diagnosed only by **proctoscope** as it is not felt by DRE. It is not reaching level of dentate line.

Second degree

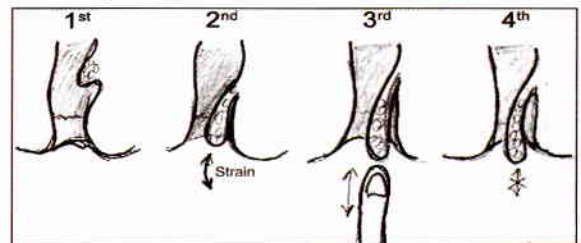
- The piles prolapse only during defecation, but they are spontaneously reduced at the end of act.

Third degree

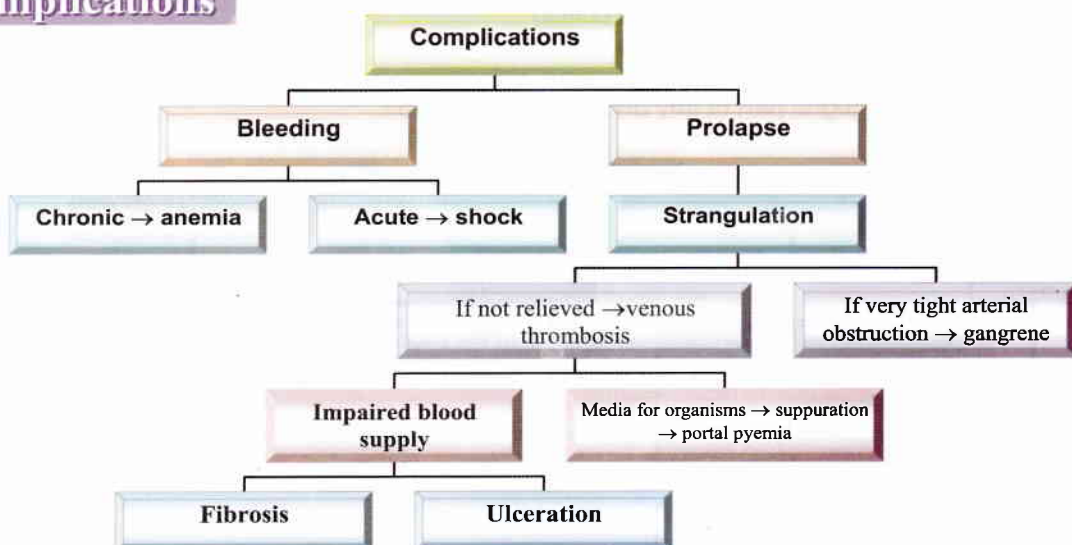
- There is prolapse of piles during defecation and patient has to reduce it manually.

Fourth degree

- There is permanent prolapse of piles.



Complications



1. Bleeding per rectum:
2. Thrombosis (thrombophlebitis of strangulated piles if not reduced within 1-2 hours)
3. Fibrosis
4. Ulceration of the mucous membrane.
5. Gangrene: if strangulation occludes arterial supply.
6. Suppuration: if secondary infection occurs → abscess, bacteremia.
7. Pyemia: (portal)
8. Partial rectal prolapse (piles are the most common cause)



9- Strangulation:

- **Pathogenesis:** strangulation of prolapsed piles by spasm of external anal sphincter (most common with 2nd & 3rd degree piles)
- **C/P:** tense, tender swelling
- **Fate:**
 - Thrombosis if not reduced within 1-2 hours.
 - Gangrene if strangulation is very tight

It is said that strangulated piles had cost Napoleon Waterloo battle and the French their empire

DD

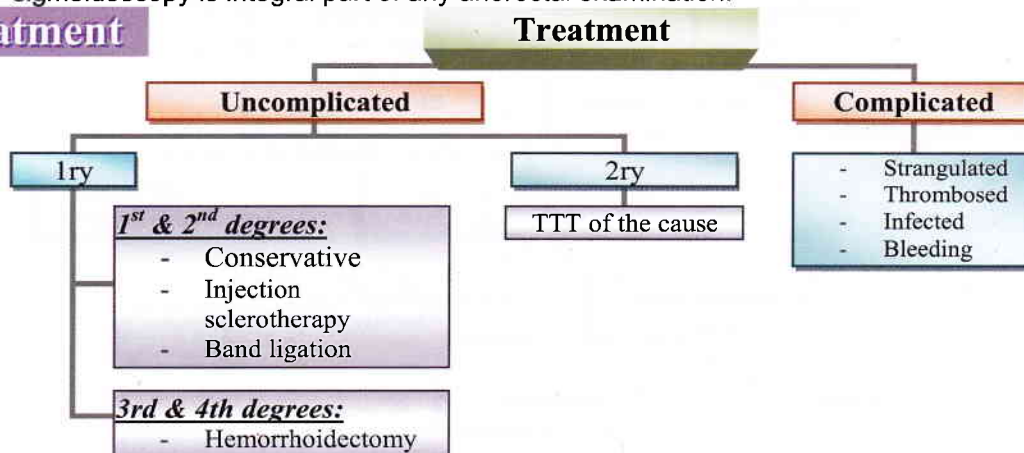
Causes of bleeding per rectum

1. Piles.
2. Diverticulum of the colon.
3. Angiomatous malformation.
4. Cancer color & rectum.
5. Ulcerative colitis.

Investigations

- **Mainly clinical diagnosis** except 1st degree by proctoscope.
- **Mainly for the cause (if 2nd piles)**
 - **Proctoscope & biopsy** (it is important to exclude cancer rectum).
 - Visualizes the internal hemorrhoids.
 - For rectal tumors & ulcerative colitis)
 - **Pelvi-abdominal U/S** (for pelvic tumors)
 - **To exclude other pathology:** barium enema, colonoscopy, CT. Rigid sigmoidoscopy is integral part of any anorectal examination.

Treatment



A- Treatment of uncomplicated cases:

Primary Hemorrhoids

➤ For first and second degrees:

- Conservative TTT:
 1. High fiber diet
 2. Small doses of laxative.
 3. Suppositories that contain decongestant.
- Injection sclerotherapy[□]:
 - Injection of irritant material (e.g. 5% phenol in almond oil) in the submucosa to induce fibrosis, obliteration of venous plexus and pulling up of the prolapsed cushions..
 - It may be complicated by pain & abscess formation.
- Rubber band ligation (good for large 2nd degree piles[□]):
 - Placing a rubber band around the piles → ischemic necrosis & separation.
- Selective haemorrhoidal artery ligation.
- Photocoagulation:
 - Raise the tissue temperature to 100°C → coagulative necrosis & separation.
- Cryosurgery (not done now):
 - Using liquid nitrogen (-196°C) → coagulative necrosis of piles & separation

➤ For third and fourth degrees:

- Hemorrhoidectomy: excision of the hypertrophied cushions with the overlying redundant skin. Healthy muco-cutaneous bridges should be left to avoid postoperative stenosis.
- Recently stapled hemorrhoidectomy allows excision without stricture.

Secondary hemorrhoids

- Treatment of the cause.

B- Treatment of complicated cases:

1. Treatment of strangulated piles:
 - If the case is diagnosed early → surgical intervention under antibiotic coverage (to avoid bleeding).
 - If the case is diagnosed late, the tissues are friable & edematous → conservative measures:

a. Rest in bed.	c. Analgesics.
b. Antibiotics.	d. Frequent warm baths.
e. Decongestive ointments (as glycerine + tannic acid) and local compresses by lead subacetate lotion [□] .	
2. Thrombosis, antibiotics, and anticoagulants.
 - If no responses → surgical evacuation of the thrombus and later on hemorrhoidectomy.
3. Infection (abscess) → drainage and antibiotics.
4. Fissure → lateral sphincterotomy.
5. Portal pyemia → antibiotics + ligation of superior rectal veins.
6. Bleeding → packs of adrenaline, later on hemorrhoidectomy.

Details of Treatment

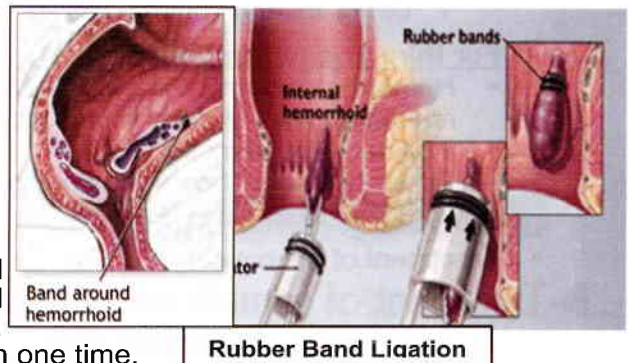
1. Injection Sclerotherapy:

- Indications:
 - Bleeding first and second degrees piles.
- The idea:
 - Is to inject an irritant material in the submucosa at the pedicle of the hemorrhoids → fibrosis → obliteration of the venous plexus and pulling up of the prolapsed cushions.
- Method:
 1. The procedure does not need anesthesia as the injection is performed in a non-sensitive area.
 2. 3 mm of 5 % phenol in almond oil is injected at the upper end of each hemorrhoid in the submucosa (not in the vessels)
 3. The procedure can be repeated once or twice after 2-6 weeks.
- Complications:
 1. Pain: if the injection is done below the dentate line.
 2. Submucous abscess formation
 3. Necrosis of the overlying mucosa.
 4. Allergic manifestations.
 5. Hematuria: if the injection is deep



2. Rubber-band Ligation:

- Indications
 - Second and early third degree piles (as it is too large for the injection)
- The idea:
 - Is to place a tight elastic rubber band around the pedicle of the hemorrhoid → ischemic necrosis and separation.
 - Better not to ligate more than two in one time.
- Disadvantages: Slippage.



3. Infrared Photocoagulation:

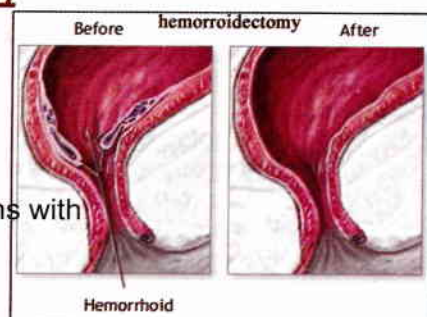
- Indications:
 - Bleeding first and second degree piles
- The idea
 - Infra-red photocoagulation can give a tissue temperature of 100°C → coagulative necrosis → separation of dead tissues after 10 – 14 days leaving an ulcer.

4. Cryosurgery: (not done now)

- The idea:
 - Apply a liquid nitrogen (-196 degree) → coagulative necrosis of the piles which separate later.
- Disadvantages:
 1. severe after pain☑
 2. Putrid discharge☑ from the anus
 3. Delayed wound healing
 4. ↑ Recurrence rate.

5. Surgical Treatment (Hemorrhoidectomy)

- Indications ☒:
 - Third and fourth degree piles.
 - Failure of non operative ttt of 2nd degree piles.
 - Fibrosed hemorrhoids.
 - Internoexternal piles.
- The idea:
 - Is to excise the hypertrophied vascular cushions with the overlying redundant skin
- The procedure: See operative.
- Postoperative:
 - Laxatives and analgesics.
- Complications:
 1. Early:
 - Pain, retention of urine (commonest ☒) and reactionary hemorrhage.
 2. Late:
 - Anal stricture ☒:
 - To avoid this complication, intact areas of anal skin and mucosa should be left between the raw areas of excised piles.
 - Recurrence:
 - Especially in young patients and is usually due to enlargement of daughter piles.
 - Injection treatment is advice.
 - One of the cutaneous wounds may not heal completely and leads to a fissure.
 - Fecal incontinence.
 - Secondary hemorrhage ☒.
 - Recently, operation is done using endostippling technique (less painful and less traumatic)



B- Acute External Piles (peri-anal hematoma)

Pathogenesis

- Rupture of a dilated anal vein secondary to straining at defecation, coughing or lifting a heavy weight → hemorrhage in the loose subcutaneous connective tissue.

Clinical Picture

Symptoms

- Sudden severe pain

Signs

- Tense, tender, bluish swelling covered by smooth shining skin

Fate

- Untreated hematoma usually resolves
- It may suppurate, ulcerate through skin or fibrose and give rise to a cutaneous tag.

Treatment

- Under local anesthesia, the hemorrhoid is bisected and the 2 halves are excised with small position of adjacent skin to leave a pear shaped wound, which is left open to granulate.



External Pile

Anal Fistula

Definition

- It is a granulomatous track which opens:
 - Internally** → into the rectum or the anal canal, usually at the level of dentate line.
 - Externally** → on the skin around the anus.

Etiology

- Infection of anal glands → abscess formation followed by:
 - Spontaneous rupture** or
 - Inadequate drainage**

Pathology

- The anal glands act as a reservoir for infection.
- The presence of an internal opening allows recurrent activation of infection
- Fecal material may act as a foreign body.
- Underlying specific diseases, e.g. Crohn's disease, TB & ulcerative colitis.

➤ So it is rarely, if ever, closes spontaneously without surgical aid.

Classifications

A. Standard classification:

- According to position of internal opening in relation to anorectal ring:**
 - Low fistula:** below the anorectal ring.
 - High fistula:** above the anorectal ring.

B. New classification (Park's classification)

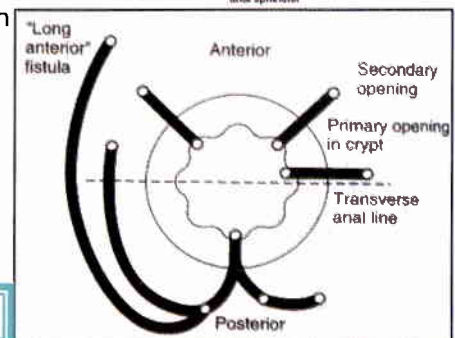
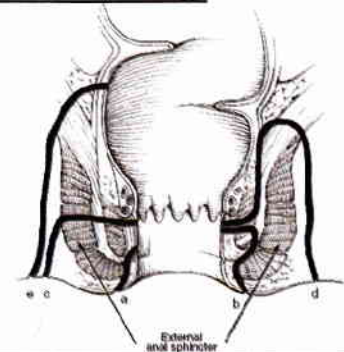
- According to the course of fistulous track:**
 - An intersphincteric fistula** tracks (most common 70%) between the internal anal sphincter and the external anal sphincter in the intersphincteric space.
 - A transsphincteric fistula** tracks from the intersphincteric space through the external anal sphincter.
 - A suprasphincteric fistula** leaves the intersphincteric space over the top of the puborectalis and penetrates the levator muscle before tracking down to the skin.
 - An extrasphincteric fistula** tracks outside of the external anal sphincter and penetrates the levator muscle into the rectum.

C. Goodsall's rule

- We can determine the position of the internal opening according to the relationship between the external opening and an imaginary line drawn between the ischial tuberosities bisecting the anus.

Goodsall's Rule

- ▶ **Etiology:** infection of anal glands
- ▶ **Class.:** Standard (high or low), Park's class., Goodsall's rule
- ▶ **C/P:** Purulent discharge, pruritis ani, pain
External opening may be seen
- ▶ **Invest.:** proctoscopy, endoluminal U/S or MRI + for the cause
- ▶ **TTT:** **A. Low fistula** → lay open (fistulotomy)
B. High fistula → - Lower part: lay open
- Upper part: Seton wire or Fibrin glue & if failed Proximal colostomy



- 1- **If external opening is posterior to transverse anal line:**
 - The fistulous track is curved (hoarse-shoe)
 - There is a common internal opening in midline posterior.
- 2- **If external opening is anterior to transverse anal line:**
 - The fistulous track is straight.
 - The internal opening is separate on the same.

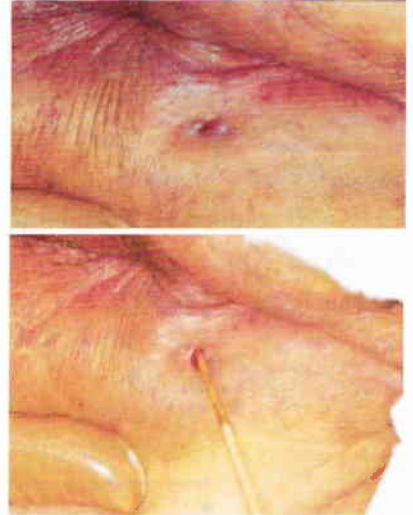
Clinical Picture

Symptoms

- History of a previous perianal abscess.
- Followed by **purulent discharge** (the most important).
- Local sourness and pruritis ani.
- Perianal pain & swelling if abscess develops.

Signs

- **Inspection:**
 - External opening may be seen as small skin elevation surrounded by granulation tissue.
 - If openings are bilateral suspect ischiorectal abscess.
- **Palpation** → Internal opening or indurated track may be felt
- **Probe examination should be delayed** and done preoperatively under anesthesia as it is painful and causes reactivation of infection and formation of new tracks.



❖ **Special types of fistulae:**

- a- Fistula in relation to fissure → pain and sentinel piles.
- b- Fistula in relation to crohn's disease → multiple with skin coloration and no skin induration.
- c- Fistula in relation to T.B. → past history of TB with multiple openings
- d- Malignant fistula in relation to colloid carcinoma.

Investigations

It is important to exclude other causes especially if multiple openings.

A-For diagnosis:

- **Fistulogram: (rarely done)**
 - With lipidol → demonstrates the track & any side branches
- **Proctoscopy:**
 - Internal opening may be seen
- **Recently:** endoluminal U/S or MRI.

B-For the cause:

- **Colonoscopy & Biopsy:**
 - From the fistula to detect cause (esp. if there are multiple fistulae)
- **Barium enema:** (rarely done if crohn's disease if suspected)
- **Chest X-ray:** if there is a possibility of TB.

DD*(Pilo-nidal sinus)*

	Pilo-nidal sinus	Peri-anal fistula
Site	near mid-line over coccyx	Close to anal verge
Probing	Upward & forward towards sacrum	Towards anal canal.

Treatment**Aim**

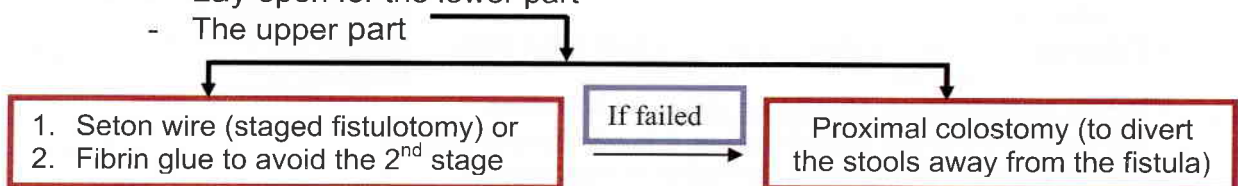
- Eradication of sepsis with preservation of anorectal function.

Options**1- Low fistula:**

- Lay open (fistulotomy[☑]) or fistulectomy.

2- High fistula:

- Lay open for the lower part
- The upper part

**Exception[☑]**

High inter-sphincteric fistula → lay open fistulotomy for both parts

1. Fistulotomy:- **General principles:**

1. The fistulous track is laid open.
2. The track is curetted.
3. Trimming of the edges, the wound is left open to heal by secondary intention.

- **Special methods of each type:**1. **Fistulotomy for an Intersphincteric fistula:**

- Division of part of the internal sphincter for deroofting of the fistulous track (no disturbance of continence mechanism).

2. **Fistulotomy for Transsphincteric fistula:**

- Division of part of the internal sphincter and superficial part of the external sphincter for deroofting the fistulous track (minor disturbance of continence mechanism)

3. **Fistulotomy for suprasphincteric fistula:**

a- 2 stage operation:

▪ **First stage:**

- Deroofing the lower part of intersphincteric component of the fistulous track by dividing the internal sphincter.

▪ **Second stage:**

- The rest of the track is laid open guided by Seton suture which may be left in place to induce fibrosis then open it.

Some surgeons inject fibrin glue in the upper part to avoid 2nd stage.

- b- Or Fistulotomy for the lower part and proximal colostomy.
- c- Fistulotomy with division of anorectal ring with primary repair → high risk of incontinence.

4. Extrasphincteric fistulae:

- Proximal colostomy and staged Fistulotomy then treat the underlying cause

2. Fistulectomy:

- Is complete excision of the fistulous track.

Anorectal Abscess

Definition

- 1ry infection of the anal gland which is present between internal & external anal sphincter.

Organism

- E. coli.

Route of infection

- Direct (where stools act as a source of infection).

Etiology

- ⇒ Bad general condition.
- ⇒ Bad hygiene.
- ⇒ Persistence of predisposing factors.

- ▶ **Etiology:** E.coli, direct infection, 1ry or 2ry
- ▶ **Class.:** Perianal (60%), Ischiorectal(30%), Submucous(5%), Pelvirectal(5%)
- ▶ **C/P:** as any abscess
- ▶ **Comp.:** as any abscess + fistula formation
So *(Don't wait for fluctuation)*
- ▶ **Invest.:** ↑ TLC, U/S, culture, fistulogram
- ▶ **TTT:** incision & drainage under G. anesthesia
TTT of the cause
TTT of complication (fistula).

➤ Primary anorectal abscess: Is due to:

1. Infection of the anal glands☐:

- The anal glands exist between the internal and external anal sphincters and open in anal sinuses at the level of dentate line.
- Infection of these glands by Gm -ve bacilli leads to the formation of an intersphincteric abscess (cryptogenic abscess) which may spread:
 - ⇒ Downwards → perianal abscess.
 - ⇒ Outwards → ischiorectal abscess.
 - ⇒ Inwards → Submucous abscess.
 - ⇒ To supralelevator space → supralelevator abscess.

- In the majority of these abscesses, there is an inner opening in the anal canal and drainage of the abscess is usually followed by a fistula.

➤ Infection of the apocrine glands or hair follicles of the perianal skin

- In such cases, the causative organisms are usually staphylococci and there is no communication with the anal canal.

➤ Secondary anorectal abscess:

1. Inflammatory bowel disease.
2. Specific infections e.g. TB.
3. Anorectal carcinoma.
4. Infection of a perianal hematoma, thrombosed piles or anal fissure.

Classification and Etiology

	Perianal (subcutaneous) abscess 60 % [□]	Ischiorectal abscess 30%	Submucous abscess 5%	Pelvirectal abscess 5%
Site	Subcutaneous close to the anus	Ischiorectal fossa	Submucosa above dentate line	Between the upper surface of levator ani and pelvic peritoneum
Etiology	• Infection of perianal hematoma. • Downward spread of intersphincteric abscess	• Lateral extension of intersphincteric abscess • Lymphatic or blood-borne infection (rare)	• Infected piles after injection • Inward spread of intrasphincteric abscess.	It is actually a pelvic abscess secondary to appendicitis, salpingitis or diverticulitis

Clinical Picture

Symptoms

- **General:** fever, headache, anorexia and malaise
- **Local:**
 - Continuous throbbing pain aggravated by defecation.
 - Painful swelling present only on perianal type.

Signs

- **General:**
 - Fever and tachycardia.
- **Local**
 - 1- Perianal: red, hot and tender swelling.
 - 2- Ischiorectal: Large indurated tender swelling filling the ischiorectal fossa. If not drained early, it will extend to the other space forming (Hoarse-shoe abscess). can be felt only by DRE.
 - 3- Submucous abscess and pelvirectal abscess: tender boggy swelling on DRE

Complications

(Don't wait for fluctuation[□])

- Otherwise, fistula will be formed. (30-60% of cases)
- Fistula is more common with enterogenic organisms e.g. E-Coli (but extremely rare with skin type organisms)

DD

Abscess connecting with pilonidal sinus.

Investigations

- 1- ↑ TLC.
- 2- U/S (if pelvi-rectal abscess).
- 3- Fistulo-gram (if fistula developed)(has little or no value).
- 4- Culture of abscess material.

Treatment

1) Treatment of the abscess

- **Urgent surgery (incision and drainage under general anesthesia).**
 1. General anesthesia
 2. Examination under anesthesia and sigmoidoscopy / colonoscopy for detection of internal fistulous opening.
 3. If perianal or ischiorectal → drainage under general anesthesia by Cruciate incision with excision of skin edges.
 4. If submucous → drainage through proctoscopy.
(If it is after injection sclerotherapy, it usually resolves spontaneously.)
 5. If pelvi-rectal → transrectal drainage.
 6. With a finger in the anorectum, careful curettage of the walls of the abscess to avoid iatrogenic fistula formation.
 7. Antibiotics are not essential except for certain situations as diabetic, immunocompromized patients, or presence of extensive cellulitis.
- The wound is packed with daily dressings.

2) Treatment for the cause

- (associated fissure) → Lat. Sphincterotomy

3) Treatment for complication (fistula)

- Low fistula → Lay open fistulectomy.
- High fistula → 2 stages operation
 1^{st} → lay open of lower part. 2^{nd} → lay open of upper part.

Presentations of common anal condition

- ➡ **Fissure:** Pain with & after defecation.
- ➡ **Uncomplicated piles:** Bleeding, prolapse.
- ➡ **Strangulated piles:** Pain & prolapse.
- ➡ **Abscess:** Pain (persistant) & fever.
- ➡ **Fistula:** Discharge.

Carcinoma of Anal Canal

Pathology

- Above the dentate line → Adenocarcinoma.
- Below the dentate line → S.C.C., malignant melanoma.

Spread

- Local & blood as usual.
- Lymphatic:
 - Above the dentate line → para-aortic LNs.
 - Below the dentate line → inguinal LNs.

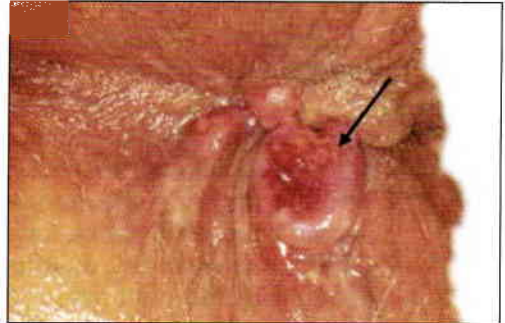
Clinical Picture

Symptoms

- Bleeding per rectum, lump.
- Pain especially with defecation.

Signs

- DRE → tumor is felt or indurated ulcer that bleeds easily.
- Inguinal LNs.



Treatment

▪ Carcinoma of the anal verge:

- Wide local excision with 2.5 cm safety margin, if there are metastatic LNs → inguinal block dissection.

▪ Carcinoma of the anal canal:

- One of two lines may be performed:
 1. Abdominoperineal dissection of the anal canal and the rectum with a terminal colostomy.
 2. Combination chemotherapy is followed by radiotherapy; the patient is examined after 4-6 weeks if there is evidence of residual tumor, abdominoperineal resection is performed

Fecal Incontinence

Definition

- Inability to retain the rectal contents at will.

Causes

- 1- **Damage to the anal sphincter mechanism due to:**
 - a- Obstetrical trauma causing complete perineal tear.
 - b- Surgical trauma: e.g. during surgery for a high anal fistula.
 - c- Accidental trauma.
- 2- **Complete rectal prolapse:**
 - The prolapsing rectum stretches the anal sphincters damaging them.
- 3- **Neurological diseases:**
 - Trauma or tumors affecting the second, third, fourth sacral nerves.
 - Diabetic neuropathies.
- 4- **Idiopathic fecal incontinence:**

Diarrhea accentuates incontinence and may even produce it in patients with border line weak sphincters

Assessment

History

- Degree of incontinence (incontinence to flatus only, to flatus and fluid stools, or to solid stools as well).
- History of trauma, surgery or difficult labor

Examination

- Rectal prolapse.
- Sphincter contraction.
- Neurological assessment.

Investigations

- 1- **Manometry:** to detect rectal functions, length and strength of anal sphincters
- 2- **Electromyography:** maps out the sphincter to detect silent areas.
- 3- **Defecography:** to assess the anorectal angle.
- 4- **Transanal U/S:** to localize the site of sphincteric damage.

Treatment

Conservative treatment

(In mild cases)

- Constipating agent and low fiber diet to thicken the stools.
- Anal sphincters and pelvic floor exercises.
- Evacuating the bowel completely with glycerin suppositories in the morning to avoid soiling later in the day.

Surgery

- Repair of divided sphincter.
- Rectopexy to restore continence in 50% of patients having complete rectal prolapse.
- Postanal repair is indicated for idiopathic incontinence.